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CORRELATIVE VALUE OF CLINICAL AND PATHOLOGICAL FINDINGS IN ROENTGEN DIAGNOSIS*

KANO IKEDA, M.D.
Saint Paul

LIKE many other methods of diagnosis in medicine, roentgen diagnosis is of relative value and cannot always be definite and final. It may be indefinite or doubtful and occasionally entirely impossible. Fortunately, thanks to the ever increasing precision in technic and to the availability of well trained roentgenologists, diagnostic difficulties have been greatly reduced. The training, experience and knowledge of the examiner, good and careful technic and sufficient clinical data all contribute materially and often decisively toward the solution of the doubtful and to even solve the apparently impossible. On the other hand, a positive diagnosis by the most experienced and careful may, at times, be found erroneous. For, after all, roentgen diagnosis depends upon the interpretation of shadows of various densities on the screen or the photographic film and not at all on the actual demonstration of the object for diagnosis. Often, at best, it represents the expression of the judgment based upon the knowledge and experience of the examiner. Herein lies the fundamental difference between the diagnosis by means of x-rays and that through pathological methods. The exactitude which may often be imposed upon the latter cannot always be expected of the former, although the precision of the present day roentgen diagnosis is often amazingly accurate and, frequently, almost uncanny in the hands of the experienced. In all cases, the best roentgen opinion would likely be obtainable when the roentgenologist, trained in clinical medicine and versed in pathological anatomy, is given the place of a consultant and the privilege of an access to all available clinical data, a careful analysis of which is often essential in the rational interpretation of the roentgen findings.

An attempt will be made to briefly present the following case reports, which have been gathered from the archives of a hospital laboratory, representing several hospital diagnostic services, in order to illustrate and to emphasize the value in diagnosis of correlation of the clinical and pathological data with roentgen findings, together with such pertinent remarks as may be indicated for further elucidation of this subject.

The lack of time necessitates the omission of all photo-micrographs from this presentation.

The cases will be divided into several groups, each illustrating a simple, fundamental, practical lesson in the broad field of medical diagnosis of which roentgenology is a major part.

Group I (illustrated by Cases 1 and 2). A very distressing experience may come to a roentgenologist when a request is made by the attending physician for a superficial or hurried examination of a patient or, as the common expression goes, "just to look at him," in order that the time, the trouble and always the expense of the examination may be minimized for the patient by this short cut method of examination. By acquiescing to such a request and literally attempting to carry it out, the roentgenologist may occasionally find himself in a most embarrassing position. It should be the policy of every roentgenologist, never to allow himself to modify his established course of technical procedure except under the most extreme circumstances.

Case 1.—E. W., male, age fifty-one. Chief complaints: Paroxysmal cough, shortness of breath and cyanosis. History of chronic gastric ulcer of twenty to forty years' standing (roentgen diagnosis of gastric ulcer

*From the Chas. T. Miller Hospital and the Amherst Wilder Dispensary, Saint Paul. Presented before the Radiological Society at the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, May 22, 1933.

eight years ago). Roentgenograms of the chest: Chronic non-tuberculous interstitial pneumonia or diffuse infiltrating type of metastatic carcinoma. Attending physician's request: "Fluoroscope his chest; give him a few swallows of barium and peek at the stomach without letting the patient know, since he absolutely refuses to have the stomach examined." Result: Negative; no plates taken. Autopsy: Lungs, diffuse metastatic carcinoma throughout. Stomach, a small carcinoma in the pyloric antrum.

Case 2.—L. H., female, age sixty-four. Chief complaints: Loss of appetite, nausea, loss of weight, weakness, fast pulse, nervousness, marked emaciation. No food distress. No palpable glands. Nodular thyroid, tremor, pulse 110. Basal metabolism, plus 35 per cent. Blood smears negative. Fluoroscopy of the stomach, without plates (by the attending physician), twice negative. Thyroidectomy. Basal metabolism remained elevated (plus 59 per cent). Autopsy: Leukemic infiltration of the stomach, the thyroid gland and other visceral organs.

Group II (illustrated by Case 3). Somewhat an identical situation may arise when the patient, found in a critical condition and unable to cooperate with the examiner, may present what may be roentgenologically considered a definite lesion, supported by the clinical findings, only to be found entirely erroneous at the autopsy table. It is well that a guarded diagnosis be rendered in those cases where the patient is found to be too ill for thorough investigation.

Case 3.—F. V., female, age sixty-four. Chief complaints: Sudden onset of complete dysphagia at breakfact; marked cyanosis and inability to talk; being treated for cardiac decompensation. Clinical diagnosis: Carcinoma of the esophagus with obstruction. Roentgen diagnosis: Apparent complete obstruction of the proximal portion of the esophagus at the level of the third and fourth cervical vertebræ.

Autopsy: Hypertension heart, 530 grams. Chronic passive congestion of the lungs and liver. Normal esophagus. No foreign body in the esophagus.

Group III (illustrated by Cases 4 and 5). Often are the moments of embarrassment and surprise to the clinician who fails to make a correct clinical diagnosis or to carefully search for the cause of the present illness, before a radical procedure is undertaken, only to resort to the belated x-ray examination after the irreparable mistake has been committed. This, again, emphasizes the imperative necessity of the most painstaking attempt to eliminate all diagnostic possibilities, by every available measure at our command in order that the patient may be spared an unnecessary radical procedure.

Case 4.—H. R., male, two years of age. Chief complaints: Abdominal enlargement began about four months ago, occasional abdominal pain, marked anorexia. No vomiting. Abdominal tap without results. Diagnosis: Celiac disease or abdominal tumor. The abdomen varied in size from time to time, was doughy in consistence, with more fullness in the right lower quadrant. X-ray of the abdomen negative. Gastro-intestinal tract negative. The mass in the pelvis displaced the cecum upward.

Laparotomy: Found what appeared to be a dilated urinary bladder. Injection of the urinary bladder during convalescence with an opaque medium revealed a marked dilatation of the ureters and renal pelvis, due probably to a congenital obstruction of the urethra.

Case 5.—Baby S., male, three weeks old. Chief complaints: Vomiting, a mass in the lower abdomen. Distention of the abdomen with prominent superficial veins. A mass palpable in the pelvis, rising up to the umbilicus; 280 c.c. of cloudy urine obtained through catheter.

Roentgen examination: Sodium iodide injection into the urinary bladder which revealed a marked redundancy and dilatation of the ureters. Post-mortem injection demonstrated a valve in the posterior urethra and a marked dilatation of the renal pelves as well as the ureters.

Autopsy: A membranous valve obstructing the prostatic urethra. A marked dilatation and tortuosity and hypertrophy of both ureters and hydro-pyonephrosis of both kidneys and pyonephritis of the right kidney.

Group IV (illustrated by Cases 6, 7, 8, 9, and 10). Not infrequently, an error in diagnosis may be committed because of the failure, justifiable or otherwise, of the clinician to locate the primary site of a malignant neoplasm. Symptoms are often only referred to the metastatic process, which, too, may not be recognized or diagnosed. It is a common and well known practice to make a clinical diagnosis of arthritis or rheumatism and so treat the patient when a roentgenogram may reveal metastatic carcinoma in the bone or a careful physical examination may elicit a small lump in the breast. Large ovarian carcinomas have been removed without first having a roentgen study of the gastro-intestinal tract made, only to discover postoperatively or at autopsy that the primary was in the stomach. Enormous, massive, metastatic carcinoma of the liver has been seen, from time to time, in which the primary proved to be an insignificant lesion in the stomach. Parallel clinical pictures in various malignant tumors have been encountered by every practitioner of medicine.

Case 6.—E. S., female, age fifty-five. First treated for arthritis of the knees without roentgenograms. Breast

cancer recognized and removed a few months later, but no search was made for skeletal metastasis. Generalized skeletal as well as visceral metastasis and death two years later.

Autopsy: Extensive cancer metastasis about the bones of the knee joints as well as all other bones.

Case 7.—M. S., female, age forty. First treated for sciatic rheumatism. Roentgenograms of the pelvis were taken but the metastatic lesions were not recognized by the clinician. Six months later, breast cancer was recognized. Generalized bony and visceral metastasis and death.

Autopsy: Generalized carcinomatosis.

Case 8.—H. Q., female, age fourteen. Admitted as a case of tuberculous spondylitis with double knuckles. Roentgenograms of the lungs were diagnosed as pulmonary tuberculosis. Roentgenograms of the spine showed a destruction and flattening of the eighth dorsal and the third lumbar vertebræ with peculiar moth-eaten rarefaction of the bones of the spine, pelvis and femurs. The grandmother then called the attention of the physician to the fact that she had received a blow on the right breast about six months before, followed by the development of a painful lump. Examination now revealed a large, irregular, hard mass in the right breast with enlargement of the axillary nodes and another mass in the left breast. Biopsy of the axillary node revealed a metastatic carcinoma. Autopsy: Generalized carcinomatous metastasis including the bony system and the lungs. Primary in the right breast.

Case 9.—M. E., female, age sixty-five. Enlarging abdomen with fluid. Palpable masses in the pelvis. Operative removal of bilateral solid ovarian tumors, microscopically diagnosed as metastatic cancer (Krukenberg type).

An x-ray examination of the stomach, during the convalescent period, revealed diffuse carcinoma involving the pyloric antrum. No autopsy.

Case 10.—M. J., female, age forty-three. Enlarged masses in the pelvis. Operative removal of tumors, diagnosed as cancer. Persistent gastric distress which was ignored by the surgeon. An x-ray examination of the stomach later revealed a filling defect at the pylorus, with retention. Autopsy confirmed the diagnosis of cancer of the pyloric portion of the stomach.

Group V (illustrated by Case 11). Sometimes, the attending physician may be so positive of the diagnosis in a given patient who has been under his care for many years for the recurring attacks of the same complaints that he neglects to resort to an x-ray examination for further enlightenment and confirmation which would be done without hesitation for another patient, presenting the similar complaints. Regardless of how easy and reasonable the clinical diagnosis may seem to be, a careful roentgen study is always indicated, if the same clinical picture persists from year to year in spite of the treatment.

Case 11.—W. S., male, age nineteen. Recurrent abdominal pain and vomiting since childhood, diagnosed as tetany and as spasmophilia. Present diagnosis, gastric tetany with persistent vomiting. Various forms of anti-spasmodic were given. Blood calcium, 7 mg. Surgical consultant requested an x-ray examination of the gastro-intestinal tract. X-ray finding: Complete obstruction at the duodeno-jejunal junction with an enormous dilatation of the duodenum. Autopsy: Volvulus of the duodenum with obstruction, on the basis of a congenital anomaly of the transverse mesocolon and of the origin of the superior mesentery and a mobile cecum and ascending colon.

Group VI (illustrated by Cases 12, 13, 14, and 15). An apparent agreement between the roentgen diagnosis and the clinical observation should not prevent the clinician from obtaining a further corroboration, either by biopsy or by autopsy. Our vital and morbidity statistics would be more reliable if the physician would obtain autopsy confirmation before the final diagnosis is rendered or the death certificate is signed. Exploration may often be justified on this ground.

Case 12.—O. M., male, age seventy-one. Presented himself in November, 1921, complaining of continuous dull pain in the stomach which had been present, then, for many years. Gastric retention with a filling defect in the duodenal cap found by x-ray.

Laparotomy showed a large mass obstructing the pylorus: enlarged regional nodes. Gastro-enterostomy was done. Diagnosis: Cancer of the stomach. Improvement. Palpable regional nodes in axilla, developed in January, 1930. Biopsy report: Leukemic lymph node. Blood picture normal. Death from cardiac failure.

Autopsy: Leukemic infiltration of the stomach, regional lymph nodes, liver, spleen and kidneys.

Case 13.—M. J., female, age seventy-one. Chronic diverticulitis of the sigmoid with obstipation and pain for six years. Palpable tumor, possibility of cancer, in the pelvis.

X-ray examination: Multiple diverticuli of the sigmoid with a partial obstruction in the sigmoid. Colostomy was done.

Autopsy: Chronic diverticulitis with perforation and abscess formation. Peri-diverticulitis. Obstruction of the sigmoid due to adhesions and kink.

Case 14.—L., male, age sixty-five. Chief complaints: Bleeding from the rectum and pain in the abdomen. X-ray examination: Multiple diverticuli in the sigmoid; no obstruction. Proctoscopic examination was negative. Diagnosis: Multiple diverticulitis. Four months later, obstruction of the sigmoid. Biopsy through proctoscope: Adenocarcinoma. No autopsy.

Case 15.—R., female, age forty-five. Diabetic. Palpable mass in the left kidney area with pain and hematuria. Pyelogram: Renal tumor of unknown type. Nephrectomy. Pathologic report: Peri-renal organized hematoma. Recovery.

Failure of the roentgenologist has resulted, not infrequently, through his failure or inability to be present at the time the roentgenograms are taken by the technician, who merely follows the instruction on the request blank. The presence of the roentgenologist is often essential in the production of diagnostic roentgenograms. The present practice of a roentgenologist directing several hospital x-ray departments, in addition to looking after his own private practice, while the best arrangement available at the present time, is not without its advantages. Under this system, there has been a tendency to place too great a responsibility upon the non-medical technician in charge in matters entirely foreign to his sphere of activity. In fact, there have been reported instances where the non-medical technician in charge (nominally under the supervision of a recognized roentgenologist) is so trained in diagnosis as well as in technic, that, in the absence of his chief, he actually makes diagnoses, writes reports, and offers expert advice to the referring clinicians who, in turn, accept such diagnoses and advice, more or less, as a matter of approved practice. All of which serve only to the detriment of the practice of roentgenology.

In conclusion, may I say that this presentation was prompted by the desire to show, by reciting instances of failure and mistake, either on the part of the clinician or the roentgenologist, that the maximum efficiency in diagnosis is obtainable only through the unreserved coöperation and interdependency between the clinician, the pathologist and the radiologist, an oft proclaimed doctrine which, nevertheless, is too frequently forgotten, alike by the attending physician and by those representing these two major specialities; and that there is a limitation, at times, in roentgenologic methods of diagnosis, the full apreciation of which may strengthen, rather than weaken, the place of roentgenology in the practice of medicine.

POSTOPERATIVE PERITONEAL ADHESIONS: CAUSES AND PREVENTION*

A. E. Benjamin, M.D.
Minneapolis

POSTOPERATIVE peritoneal adhesions are the *bete noir* of the abdominal surgeon. Nevertheless, if it were not for the fact that nature rallies to the defense of the organs within the abdominal cavity by the formation of adhesions, greater functional damage to these organs would occasionally occur (Fig. 1).

Primary adhesions usually follow some disease occurring in or around:

(1) The gallbladder and liver, from stones or infection; (2) stomach and duodenum, from ulcer or cancer; (3) appendix because of infection (Fig. 2); (4) intestine, colon and rectum, from inflammation, diverticulitis, cancer or tuberculosis; (5) bladder, from cystitis; (6) uterus, tubes and ovaries, from infection, new growth and pressure. Adhesions when found associated with these organs may have prevented microbic invasion of the peritoneal cavity and a general peritonitis.

In performing a laparotomy upon a patient in whom a previous abdominal section has been made, adhesions will often be found, the omentum or loops of the bowel being glued together or to the abdominal wall, uterus, ovary, bladder, sigmoid, or rectum. No doubt many of these could have been prevented had a more thorough primary operation been performed, with eradication of all of the infected tissue and the careful exclusion of the traumatised area from the pressure of contiguous infected intestine by peritonealizing these areas.

It is not unusual for physicians to attribute symptoms which cannot be traced to any actual disease within the abdomen to adhesions, especially when the person has had a previous laparotomy.

In an article written several years ago on "Intestinal Adhesions" the author stated that adhesions often follow laparotomies from the use of antiseptics within the abdomen, unnecessary

*Read before the Minnesota Academy of Medicine, March 8, 1933.

handling, denuded areas, hemorrhage, and infection.

There are many surgeons who are of the opinion that there is a special tendency on the part of some individuals to develop postoperative peritoneal adhesions while in others there is a proteolytic enzyme that seems more active in digesting the adhesions.

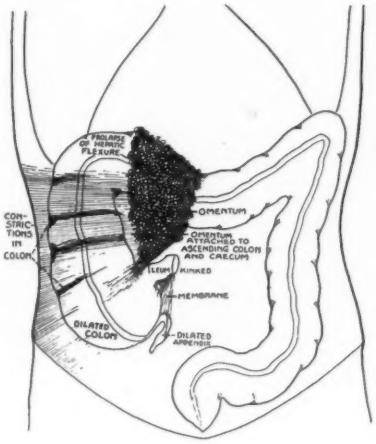

Fig. 1.

Ochsner and Garside[13] believe that a proteolytic ferment is liberated from the polymorphonuclear cells which digests the adhesions when they have served their purpose, but that in some individuals there is an inherent tendency toward the development of fibrous tissue—an "adhesion diathesis" and "keloid tendency" in which the adhesions are apt to re-form. From certain experiments they found that if saline was put in the abdominal cavity after the separation of adhesions, few or no adhesions would form in 13.32 per cent of cases and if trypsin or papain solution were added few or no adhesions were formed in 42.28 per cent and 90.89 per cent, respectively.

Kelly and Noble[8] state that certain individuals have a tendency to develop these adhesions as some do keloids.

Ladwig[10] does not think that trauma alone is responsible for adhesions and that the phagocytic action of proteolytic enzymes is responsible for the absorption of adhesions. Is it not possible that this proteolytic enzyme may absorb adhesions within the abdominal cavity unless there still remains some infection such as that residing in a loop of intestine that continues the infectious process and perpetuates the adhesions?

Haberland[5] says "it is difficult to prove that there is a constitutional disposition towards the formation of abdominal adhesions," but it is often an excuse of the surgeon for adhesions following operations.

Fig. 2 *(left)*. Adhesions around appendix because of infection.
Fig. 3 *(right)*. So-called Jackson's veil. Note double gunshot barrel arrangement of ascending and transverse colon.

The opinion was expressed by the author in an article[2] published in 1911 that the use of non-absorbable or chemically treated suture material, especially if left exposed in the abdomen, is responsible for adhesions. According to experiments, silk or linen sutures caused the most adhesions, chromic catgut was next, but when plain catgut was used alone or to cover the other sutures and careful technic was employed, few adhesions resulted. The non-absorbable sutures sooner or later found their way into the intestinal canal, but the adhesions left were firm and fibrous and persisted.

Mueller and Rademacher[12] conducted some important experiments on guinea pigs and found that when an abdominal incision was packed with sterile gauze, no adhesions resulted in any of the ten cases. Adhesion resulted in one case in ten when the wound was packed with gauze soaked in 5 per cent iodine, while adhesions occurred in six out of ten cases when the wound was packed with gauze soaked for twenty-four hours in a culture of B. coli, the adhesions extending into the muscles of the bowel.

These experiments show definitely that infection will travel through the peritoneum to the abdominal cavity and involve the contiguous tissue. We know it will travel through the walls

of a diseased appendix and involve the surrounding structures. An abdominal abscess may destroy the parietal peritoneum and even find its way to the skin surface. Infection will travel through any portion of the wall of the intestine when the peristaltic action is long interfered with.

We have all observed the so-called Jackson's veil, a thin vascular membrane on the ascending colon and cecum. A mild form of this membrane has been found in the newborn. Could not such a membrane present in infancy be the primary cause of persistent constipation and stasis, and later result in a more definite disabling membrane? A pronounced form of this membrane has been found involving the cecum, appendix, ascending and sometimes the transverse colon. Occasionally it might constrict certain regions of the colon and even cause obstruction. The hepatic flexure would be accentuated, and at times a double gunshot barrel arrangement of the ascending and the transverse colon would be seen (Fig. 3). Sometimes the cecum as well as the ascending colon would be greatly dilated.

The origin of this inflammatory reaction evidently comes from within the colon and no doubt these membranes or adhesions are protective in nature. The greater the constriction because of the bands, the more the stasis and the more infectious are the contents of the bowel, and the greater the inflammatory reaction without the colon, necessarily then, the greater the number of adhesions following—establishing a vicious circle. Should a laparotomy be performed upon a person in such a state postoperative adhesions are inevitable.

If, to intestinal infection, are added ingested microörganisms from diseased tonsils, teeth or sinuses or those gaining entrance from a diseased gallbladder, the infection within the alimentary canal is greatly augmented, increased inflammatory processes occur and peritoneal adhesions follow, especially after a laparotomy.

I have a number of patients who have had these protective adhesions interfere considerably with the function of the bowels. Some were not cured until all foci of infection, *i.e.*, infected teeth, tonsils and sinuses, were cleaned up. From this experience I have concluded that such foci have much to do with the cause of the perpetuation of peritoneal postoperative adhesions.

No doubt there are many causes for postoperative peritoneal adhesions, any one or more of which may be responsible in an individual case. It is difficult to demonstrate that the fault lies in the lack of a proteolytic enzyme in the patient that is capable of digesting adhesions.

Time should be taken to get the patient in good condition for operation unless an emergency exists. The intestinal tract and all organs directly connected with the function of or in conatct with the alimentary canal should be investigated. The normal tone and activity of the bowels, especially of the colon, should be attained, if possible, before the operation is performed.

The influence of infectious foci on the health of the gastro-intestinal tract and their relation to pericolic adhesions has been related in a former article by the writer.[4] I am convinced that to get the best results and avoid adhesions is to negate that influence

An anesthetic should be given so that there is complete relaxation of the abdominal wall and no harm done to the peritoneum from straining, coughing, or vomiting.

The skin must be thoroughly cleansed but the intestines should not come in contact with any irritating chemical on the skin while operating. Strong chemicals may destroy the endothelial cells and promote adhesions.

The position of the patient should be such that the abdominal muscles are relaxed and the intestines gravitating away from the field of operation.

The incision should be over the most convenient portion to get at the diseased area, avoiding injury to the nerve supply, and sufficiently large for good exposure and complete, careful work.

Powder or chemical solutions on gloves are irritating to the peritoneum. The gloves should be thoroughly washed with sterile water before operating and should be changed at once if punctured or torn.

Dry gauze packs are irritating, warm moist ones are less so, but hot ones are very harmful. Undue crowding of intestines against the pack while the patient is straining or vomiting may injure the peritoneum. Also denudation may occur if the packing is removed roughly.

Percy[14] holds up the edges of the wound with the gloved hand while packing.

A rubber sheet for packing is less irritating, prevents the loss of heat and exposure to air and

is cleaner. Gauze may leave particles of lint which damage the peritoneum and invite adhesions.

Warm wet sponges should be used and rubbing avoided.

Long exposure of the peritoneum to heat, light, and air is harmful. The peritoneum soon dries out and the cells may not readily recover.

Prolonged and firm automatic retraction with wire or narrow bladed retractors, or scraping the peritoneum with rough hand retractors causes cell destruction and therefore should be interdicted.

Care should be observed to avoid pressing within the abdomen with instruments or grasping the intestines or peritoneum, especially at the wound margins, with instruments. The ready threaded atraumatic needles are best used for suturing intestines or peritoneum, as we know the peritoneum may be torn when the usual large eye needles are used. The intestine may become fixed at that point and infect the area thus traumatised.

It is best to avoid unnecessary separation of adhesions around an infected area. To wall off well and evacuate pus by using the suction apparatus is a safer practice. If the operation can be performed when the inflammatory process is subsiding there is less chance of extension with multiple adhesions following.

In performing a salpingectomy all of the infected tissue should be removed by dissecting out the corneal portion and covering this area well with the broad and round ligaments. Adhesions can also form around a long appendiceal stump or a portion of an infected ovarian cyst.

We should correct associated diseases within the abdomen when possible. When it is necessary to correct a displacement of the uterus it is wise to remove an appendix if there is any sign of trouble present. When a disabling membranous pericolitis exists with a diseased appendix, it should be attacked simultaneously unless the appendicitis is too acute.

Irritating chemicals are harmful. The peritoneum is less resistant than the infectious microorganisms; therefore, the chemicals are worse than useless as they may do irreparable harm to the peritoneum and promote adhesions.

Healthy contiguous peritoneal tissues do not form adhesions. When raw, traumatised, or chemically treated areas and non-absorbable suture lines are covered by transplantation of a contiguous healthy peritoneum or omentum, harmful adhesions are not likely to occur (Fig. 4). This method is always practiced by Baldwin.[1]

It is best to cut adhesions instead of rubbing

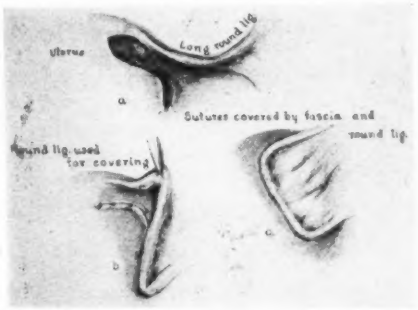

Fig. 4. Peritonization following suspension of the uterus, combined with removal of the tube and ovary.

them off and to cover and turn in all the cut edges with a fine plain catgut suture. Adhesions around tuberculous or malignant disease are best separated by the use of the diathermy cautery. A fairly deep cauterization leaves fewer adhesions than a superficial one and is more suitable than the knife for removing the disease.

In case of inevitable adhesions the viscera should be replaced where the functions will be best performed.

Blood is a very good culture medium. Clots left in the abdomen favor adhesions when they become infected. All bleeding vessels should be secured by catgut ligature.

It is best to drain infected areas adequately with Penrose drains assisted by gravity when possible, namely, by turning the patient onto the abdomen where abdominal abscesses are present. These drains are less harmful than pus retained under pressure. Without drainage diffuse peritonitis is possible. The pus may be forced into other areas and a wide range of adhesions follow. We should avoid gauze drains and undue pressure from firm rubber drainage tubes.

Chemically treated suture material and nonabsorbable sutures should not be used in abdominal operations unless peritonealization is employed, using fine plain catgut for the work. The outer suture of the intestine should be of plain catgut with an atraumatic needle (Fig. 5).

The intra-abdominal use of saline solution is considered by most surgeons an aid in preventing adhesions. It is of doubtful value, but may do no harm.

Vaseline and oil have been used with the hope of temporarily preventing adhesions between

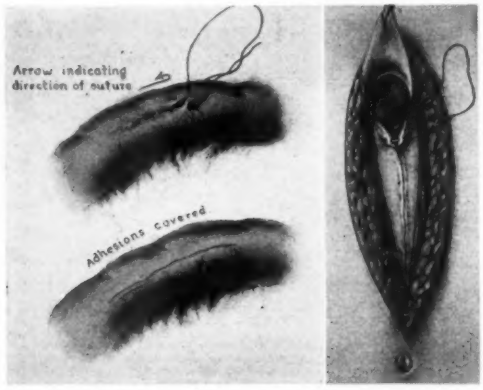

Fig. 5 *(left)*. Technic for burying knots in peritonization.
Fig. 6 *(right)*. Proper method of peritoneal closure.

raw, denuded, and traumatised areas until nature has safely covered these spots with a healthy growth of cells. I am certain that, when used sparingly, simply spreading a thin film-like coat of sterile vaseline over extensive uncovered raw areas, it is of some benefit. It lessens the bleeding and exudate and has in no case in which I have used it done any harm, but on the contrary it has probably assisted the healing process underneath this film by keeping the contiguous loops apart.

Air has been forced into the abdominal cavity for the purpose of keeping affected tissues apart until normal peritonealization has taken place. Is it not likely that this will increase the intra-abdominal tension and the possibility of adhesions? An injured intestinal wall soon becomes thickened and edematous, and, the healthy portion being more easily compressed, the swollen areas would be forced into closer contact.

Experiments by Ochsner and Garside[13] seem to prove that trypsin and papain lessen the number of adhesions materially.

The fact is mentioned in Keen's Surgery[7] that antifibrin ferment is beneficial as it prevents the coagulation of the exuded serum.

Johnson,[6] from a number of experiments, concluded that amniotic fluid is safe to use in the abdomen of the human being. He states that this fluid should be reserved for clean cases and the effectiveness is conservatively estimated to be 70 per cent.

Lacey[9] in his last report concludes that:

1. "Amniotic fluid is apparently harmless when introduced into the peritoneal cavity.
2. "It would seem to lessen the ooze from denuded surfaces and stimulate the peritoneum to a more powerful defense reaction.
3. "It cannot be depended upon to prevent adhesions, although it possibly does modify their density."

Mann[11] in a personal communication states: "I am not sure as a result of our experimental work whether it would be of sufficient value for clinical use."

I have used amniotic fluid in an endeavor to prevent adhesions, but I am not convinced that it has any value.

In a paper read before the St. Louis County Medical Society in 1913 on "Intra-abdominal Complications Following Laparotomies,"[3] I called attention to the fact that intra-abdominal tension was responsible for spreading infection and promoting adhesions.

It has been my experience that cases in which there has been a great deal of distention of the abdomen with vomiting following a laparotomy and where it has been difficult to get a bowel action that the patients seemed to develop symptoms later of adhesions. Great intra-abdominal pressure must force the intestines and omentum firmly against the peritoneal incision or other areas that may have been traumatised at operation. This firm pressure cannot but favor adhesions in these areas. If we can lessen this intra-abdominal distention in any way, I believe adhesions are less likely to occur.

Change of position, except in acute infection, even turning the patient onto the abdomen, makes it less likely for gasses and contents to be trapped in coils of intestines. The use of the rectal tube occasionally will relieve the accumulation of gas in the lower bowel above a firmly contracted sphincter.

Intestinal stasis from kinks and obstruction causes reverse peristalsis and the contents coming into contact with a mucous membrane unaccustomed to these bacteria produces toxic effect. The patient is unable long to endure such a state. It is apparent therefore, that the fecal

current must be kept moving downward for the patient's welfare. The longer this state of affairs exists, the greater the disturbance, the slower the convalescence and the possibility of adhesions is increased. The extent and firmness of the adhesions may be in direct ratio to the virulence of the infection and the degree of the tension.

The most useful agency for removing gas and thereby preventing gastric dilatation is the nasal suction tube. It allows the intake of quantities of cooling fluids by mouth, which lowers the temperature, stops vomiting, carries off regurgitated poisonous substances within the bowel and promotes an early normal function of the gastro-intestinal tract.

When adhesions that are slowly obstructing the bowel are separated, the fecal current becomes more normal, imprisoned infectious contents are liberated and passed out of the obstructed loop to which adhesions may be fixed. These adhesions may not recur because the contents of the bowel are restored to normal and not infectious, which is the reason, no doubt, why we obtain cures in some of the cases.

In closing the abdominal wound the peritoneum should be turned outward and closed securely with plain catgut, no raw or traumatised areas being left within the abdomen (Fig. 6). The posterior sheath of the rectus should also be sutured with chromic catgut supplemented by stay sutures; the skin closed with subcuticular dermal, clips or plain sutures. An imperfect closure of the peritoneum and fascia invites hernia and adhesions. An insecure closure may result in the bowels wedging through the opening especially if a suture breaks. Peritonitis, obstruction and adhesions may result.

Tight suturing may cause necrosis, may produce an exudate, and can result in stitch abscess or wound infection. This infection of the abdominal wound may extend to the peritoneum; therefore, it should be drained early to prevent extension into the abdominal cavity.

Suture drainage or the use of a small Penrose drain is essential in case of oozing of a wound, to prevent the presence of a good culture medium for infection. All bleeding vessels should be tied to prevent unnecessary exudate in the wound area.

A change of position of the patient favors peristaltic action. The head and foot of the bed should be elevated alternately and the patient turned from side to side occasionally in non-infected cases. The gravitation of pus in the prone position favors the early healing of the wound.

Distention of the bladder should not be allowed, especially in the presence of a cystitis, as infection seems to travel through the viscus, causing adhesions.

Following an operation water with some alkali added is best tolerated at first and may be given in large quantities early, when the nasal suction tube is used. Strained liquids may be given soon; then oily laxatives in small quantities to assist in restoring the peristaltic activity of the bowels.

CONCLUSIONS

1. The patient should be well fortified for a laparotomy by diet, exercise, and necessary medication.

2. The functions of all the organs and alimentary canal should approach normal: a colitis treated, and infectious foci eliminated.

3. The incision should be adequate and cause little injury to the nerve supply.

4. The abdominal wall should be thoroughly relaxed during the operation. The peritoneum should receive a minimum exposure to light, heat, and air. A rubber sheet is best used for packing as it does not irritate, conserves heat and moisture, and, unlike gauze, leaves no lint in the abdomen.

5. The endothelial cells should not come in contact with irritating chemicals nor powder on the gloves.

6. Retractors should be used with care, and sponging done gently.

7. All operative work should be as thorough as possible, consistent with safety, leaving no associated diseased or infected tissue nor uncovered injured areas to invite adhesions.

8. Abscesses should be completely walled off, emptied by suction, and adequately drained.

9. Solutions, fluids, oils, and enzymes are of doubtful preventative value and should not be depended upon.

10. The abdominal wall should be carefully and securely closed, with no undue suture tension, leaving no gaps or traumatised areas inside the peritoneal suture line.

11. Excessive intra-abdominal and wound suture tension is avoided, infection circum-

scribed, and fluids tolerated immediately by instituting the continuous nasal tube suction.

12. The change of position in clea cases and the cautious administration of food aid uninterrupted recovery and help prevent permanent adhesions.

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DISCUSSION

Dr. A. A. Zierold (Minneapolis): This should be an extremely fertile field for discussion. Perhaps no part of surgery excepting intestinal obstruction, or the question as to whether or not we should remove a chronic appendix, or whether or not to remove or drain a gallbladder, has been given so much attention as this question of adhesions.

I assume from what Dr. Benjamin has shown us that he has included in his remarks all the developmental peritoneal folds and attachments incident to the variations of normal position and attachment that may occur in all of the viscera. If one considers these as adhesions susceptible to surgical treatment then the field of surgery becomes at once unlimited. To attempt to explain such adhesions on pathological grounds is most difficult. I believe the most satisfactory theory is that of Sir Arbuthnot Lane, who stated that adhesive bands giving rise to the "Lane kink" and other so-called adhesion deformities were due to "crystallization of lines of force." This absolutely precludes any further discussion of the matter on rational grounds. It is probable that he as well as many who follow him in this country, notably Coffey and others, have taken exception to the limits of normal variation in the attachment of the abdominal viscera. Abdominal surgery owes its progress to the formation of adhesions. If it were not for peritoneal adhesions we could not perform operations upon the intestinal tract. Undoubtedly many adhesions occur which are not fortuitous, but I believe that we can confine the term adhesion as symptom-producing to those adhesions which produce obstruction, either total or incomplete. Why these should occur, there is apparently no adequate explanation. It is probable that the process goes through a very definite cycle of inflammatory reaction. There is no question but that every abdominal operation is followed by intra-abdominal adhesions of one sort or another. Certainly they are not all extensive or symptom-producing. I believe that the comment of Dr. Charles Mayo relative to adhesions is still pertinent and sound, *i.e.*, "Adhesions between movable viscera or movable parts of abdominal viscera are of little consequence, but adhesions between abdominal viscera and a fixed point are sources of danger."

Dr. O. H. Wangensteen (University): I fully share Dr. Zierold's attitude of mind concerning the surgery of adhesions. Apart from the few instances in which operation may be necessary for the relief of pain, when adhesions acquire attachment to the parietal peritoneum and exert traction upon it and consequently give rise to distress, the only operative indication for adhesions generally agreed upon by surgeons is intestinal obstruction.

The illustrations which Dr. Benjamin has shown us are reminiscent of the discussions frequently heard a decade or more ago when it was ardently debated whether the pericolonic membranes fixing the cecum should be divided and what should be done with the cecum in which peritoneal fusion was defective. In 1922 when the Western Surgical Association met in Minneapolis, Dr. Coffey, of Portland, read a paper on "The Significance of Right-sided Abdominal Pain," which dealt chiefly with these abnormal membranes and kinks about the cecum. Dr. Eisendrath pointed out that these abnormal folds were as frequent in the left colon as on the right side. The discussion which followed generated more heat than light.

In speaking of Mr. Arbuthnot Lane's surgical treatment of these bands, kinks and folds about the cecum, one of the surgeons at St. Mark's Hospital in London related to me some years ago a story told at the Royal Society of Medicine where the subject was under dis-

cussion. The speaker, my informant told me, said that he had "often seen Mr. Lane do these operations and it reminded him very much of a squirrel running up and down a tree. The consummate grace and beauty with which he did it aroused his admiration but why in hell he did it, he probably would never know."

The formation of intra-peritoneal adhesions is but a part of the normal process of repair and the problem is not so much one of prevention but rather an attempt at limiting this repair process during the healing stage to the injured endothelial membranes.

Of the large number of substances that have been recommended to prevent the formation of adhesions and have been given adequate clinical trial, very few have stood the test of time. There are essentially four types of agents that have been employed to obviate intraperitoneal adhesion formation: (1) the application of greasy or oily substances over the denuded peritoneal surfaces, such as liquid paraffin, vaseline or acacia. The testimony of experimental evidence would indicate that these remedies stimulate rather than prevent adhesion formation; (2) the administration of drugs such as eserine and pituitary extract to augment intestinal activity in the immediate postoperative period; (3) introduction of dilute solutions of tissue digestants such as pepsin or trypsin into the peritoneal cavity; (4) the use of fluids which are slowly absorbed from the peritoneal cavity. It is well known that in the ascitic variety of tuberculous peritonitis adhesions are rarely seen. Saline and sodium citrate solutions are absorbed too quickly to be of any value in floating the intestinal coils apart from one another during the healing period. Amniotic fluid has been used for this purpose with varying results by a number of investigators. Blood taken from the patient prior to operation and defibrinated may prove to be of some value in the prevention of inter-intestinal adhesions.

Adherence of the intestine to the anterior abdominal wall can be prevented by keeping the intestine separated from the anterior abdominal wall during the immediate postoperative period. Air may be introduced into the peritoneal cavity on completion of the operation, and, with the patient's buttocks elevated on a couple of pillows, the air will accumulate between the intestinal coils and the anterior abdominal wall, in the vicinity of the umbilicus. For a patient who had previously been operated upon thirty times for intestinal obstruction of an adhesive character during the five-year period immediately preceding, I did the thirty-first but my first operation about two years ago. A complete lysis of the small intestine was done and on completion of the operation 1,000 c.c. of air was put into the peritoneal cavity and the patient's buttocks were elevated as has been described. I have had no opportunity to ascertain whether she still has a free peritoneal space, but no more operations have been necessary. During the two-year interval I have seen her three times for mild attacks of abdominal pain. On none of these occasions has she appeared to have intestinal colic. Each time a film has been made of the abdomen and no gas has been visualized in the small intestine. On each occasion, the pain has subsided after brief treatment with conservative measures. On her first return, the trouble seemed to be an acute salpingitis; no surgical interference has been necessary. I have done this procedure in a few other patients with universal adhesions of the intestine, who have had frequent attacks of acute obstruction. In all of the other cases, however, the results have not been as striking as in the instance related above.

The method I do believe is of value in the prevention of adhesions to the anterior abdominal wall. The difficulty of keeping the intestinal coils separated from one another, however, is readily apparent and is a measure of the difficulty in preventing inter-intestinal adhesion formation.

DR. BENJAMIN (closing): I am in perfect accord with what Dr. Zierold and Dr. Wangensteen have said. I did not show these pictures to mean that all these adhesions should be operated on. We know that adhesions are protective in nature and if it were not for adhesions which protect the peritoneal cavity we would get a general peritonitis and death of the individual in infected cases. It is only when we have severe pain or symptoms of obstruction that we operate.

But why do some have adhesions with obstruction and others not? I am trying to show there are some adhesions that could be prevented. If you watch some surgeons operate you can tell that they are going to get adhesions. It is not just one thing that you do, but I have enumerated many things that must be kept in mind and if you omit one or more of these you may get adhesions. I have operated upon a number of patients for adhesions and was surprised at the results where there were adhesions of loops on intestines. Why do they adhere to one another? The point I was trying to make was that a great many of these adhesions come from infection within the bowel. By liberating the loops the intestine empties itself, becomes active, and adhesions may not re-form. Preventing intra-abdominal tension after the operation and keeping the abdomen flat helps a great deal. If there is air in the stomach it presses the loops of the bowel together and adhesions are likely to form. If you can prevent that and get a fecal current flowing soon after operation you are not liable to get adhesions.

It is not necessary to have as many adhesions as we do today. We believe some of these membranes are natural, as investigators have found them in the newborn. The ascending colon may be in the wrong position from imperfect rotation. The congenital membranes perhaps cause no trouble for years, but when one gets obstruction, of course it is necessary to operate on them. The reason we get a cure is that we liberate the infection in the bowel. In nine cases out of ten of pronounced constipation there is some malposition of the intestine or some adhesions. If the patient is well otherwise, I have found no case but what had normal action after operation and stayed that way until adhesions re-formed.

SURGICAL TREATMENT OF CONGENITAL CLUB-FEET*

GEORGE A. WILLIAMSON, M.D.
Saint Paul

ANY discussion of the operative treatment of congenital club-feet should be prefaced by a general outline of the proper conservative treatment to be used before considering any surgical procedure.

It does not come within the scope of this paper to discuss the etiology or pathology of the condition except where it bears directly upon indications for surgical procedures.

One is apt to feel, after reading a number of papers describing the conservative or manipulative treatment of this condition, that a good result is easily obtained if one persists long enough along conservative lines. So many factors have bearing on the ultimate outcome in each case that the picture is only too often an entirely different one than that so frequently painted.

It is not our purpose to enter into any argument relative to the advisability of conservative in preference to the surgical treatment as that point is conclusively settled in the minds of all orthopedic surgeons and is here reiterated. No case of congenital club-feet should be subjected to operative treatment until the maximum degree of correction has been obtained by conservative means. If treatment can be started when the child is a week or two old, surgical intervention will seldom be required. It is in the resistant, relapsed or neglected case that we are primarily interested.

One meets with an occasional early case which resists treatment of the best type begun at once and carried on through an adequate period of observation. The portion of the foot giving the most trouble is usually the talus and calcaneus. A contracted calf muscle or thickened and contracted posterior ankle-joint capsule keeps the calcaneus pulled proximally in the leg so that the heel is deficient or poorly formed. In addition, the calcaneus and talus are frequently held in a position of inversion on the tibia, which means that weight-bearing will come through the outer and inferior surface of the heel bone. The result is a tendency for the entire foot to roll into inversion and adduction with each step. Painful callosities and bursæ develop as the child walks and becomes heavier.

The typical deformity of a congenital club-foot is one of (a) inversion of the entire foot so that the lateral surface becomes the inferior; (b) adduction of the front portion of the foot at the mid-tarsal region; and (c) equinus, which means that the heel is drawn proximally and the front of the foot points distally. In correction of these deformities, as has been pointed out in many articles, the adduction of the forefoot should be overcome first of all. After the long axis has been straightened, the inversion is overcome, bringing the foot directly under the tibia in a good weight-bearing position. When this has been accomplished the foot forms an excellent lever with the ankle joint as a fulcrum for the stretching of the components of the calf. Other typical deformities develop in untreated cases as weight-bearing is begun. These are chiefly the result of the weight of the body on the deformed feet although undoubtedly the pull of strong muscles has much to do with increasing the degree of deformity. Callosities and bursæ develop over weight-bearing points and are frequently painful, particularly as the child gets older. Internal torsion in the shaft of the tibia results in a pigeon-toe deformity apparent even after the club-foot is corrected. Knock-knee proves to be very troublesome in other cases.

As has been stated before, if treatment is begun when the infant is but a week old very encouraging results can be obtained from the gentlest manipulations, aided by retentive apparatus in the form of frequently changed plaster or adjustable metal splints. It has been disappointing, however, to find in occasional cases that the foot is apparently corrected very well but on closer examination the calcaneus proves to be held drawn up in the leg and rolled into varus by resistant contracture of the calf muscle. These are particularly disappointing deformities and are very resistant to all conservative treatment.

Cases which have been well corrected by con-

*From the Department of Orthopedic Surgery, The Miller Clinic, Saint Paul. Read before the Minnesota Orthopedic Society as part of the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, May 24, 1933.

servative means require careful observation over a period of several years to guard against the recurrence of deformity. Here again one is frequently disappointed by the way varus and equinus develop due to insufficient recovery of power in the peroneal muscles, too short a period of protection of the foot in splints, plaster or braces, or failure to correct the inward twisting of the shaft of the tibia. Many authors tell of the difficulties of treatment in these so-called relapsed cases and this has been borne out in our observations.

In addition to the early case which has never been completely corrected and the relapsed case we have neglected club-feet. Although there is no reason why any individual with a club-foot deformity should be neglected it is only too often in orthopedic clinics or hospitals that one hears about how the parents were told to wait until the child was a little older or that it would grow out of the deformity. We must continue to make a plea for early vigorous treatment in all cases of congenital club-foot. Fiske reports 200 consecutive cases in the Children's Hospital of Boston in which 95 per cent of good or fair results were obtained merely by manipulative procedures.

Fig. 1. Unilateral club-foot in a boy four and one-half years of age, inadequately treated by conservative means after reaching age of one year. Correction obtained by subcutaneous plantar fasciotomy and wrenching.

Fig. 2. Untreated bilateral club-foot in a boy eight years of age. Correction obtained by forcible manipulations, fasciotomy and, later, posterior capsulotomies and removal of wedges of bone from the tarsus.

It is not our purpose in this paper to consider conservative treatment as this part of the subject has been covered in voluminous writings. The surgical treatment has not been described to any great extent.

Treatment of Infants.—In infants there is very little need for cutting in order to obtain complete correction. It is not until the child is at least nine months old that it is to be considered. In our experience about all that is needed from operation is a lengthening of the heel cord by subcutaneous incision, by an open operation or, in addition to lengthening the heel cord, a division of the posterior capsule of the ankle joint, allowing the calcaneus to rotate distally into its normal position. As is often the case at operation the Achilles tendon is found adherent to its sheath as a result of the conservative

manipulations resulting in an impairment of function. The question arises as to why it would not be better to obtain a correction of the deformity by easy manipulation after a simple operation with sharp dissection rather than obtaining a gradual correction by much force and prolonged fixation which lead to formation of adhesions. In each case adhesions are formed but impairment of function is not a prominent feature. Following lengthening of the heel cord alone or with division of the posterior capsule, a plaster of Paris dressing is applied and left on for about six weeks, after the removal of which physiotherapy and further retention in metal splints are used. It is the aim in both conservative and surgical treatment to obtain correction of the deformity by putting the foot in abduction, eversion and dorsiflexion.

Treatment of Older Children and Relapsed Cases.—In older children who have had treatment but in whom the deformity has not been entirely corrected or has been allowed to relapse, the treatment must be more radical than that mentioned previously. The deformity in these cases may be a complete return to adduction, inversion, and plantar flexion of the foot, made worse because of the fibrosis which has occurred in joint capsules and in ligaments as a result of the previous conservative course of treatment. It may be adduction, inversion and equinus of the calcaneus so that the heel does not come to the ground in good weight-bearing position. It may be simply a pigeon-toe deformity due to an internal rotation in the shaft of the tibia although the foot is completely corrected. If allowed to persist as growth goes on the bones develop in the deformed position and as a result assume abnormal shapes, making complete correction by conservative means impossible. We have here, therefore, another reason for early and complete correction of the primary deformity, and in this part of the paper consideration of the deformities due to osseous changes will be left to the neglected or older cases. We have had occasion to do considerable work on the second type of deformity, namely the adduction inversion and equinus involving the heel, and more will be said of this later.

Before any cutting operation is done on these types of deformities the full degree of correction which can be obtained by more conservative means must be assured. Manipulation of the foot under anesthesia with the assistance of the Thomas wrench or levers such as have been described by Hauser, Bettman, Telson and others in many cases yields a surprising amount of correction. It is only after a period of confinement in plaster of Paris, following a forcible manipulation, that one should consider radical treatment.

Surgical measures after removal of the plaster usually are confined to the soft parts. A contracted Achilles tendon may be lengthened by subcutaneous incision or by tenoplasty through an open incision, using any one of the many methods which are described. It must be remembered that with each lengthening of the tendon the muscle belly of the calf shortens and is made weaker. Too enthusiastic or repeated division of the contracted heel-cord frequently results in the calf muscle being represented by a small round mass just below the knee contracting weakly because of the shortened excursion of its fibers. Carefully chosen cases, however, are benefited by the correction of the equinus deformity.

Even with the lengthening of a tight Achilles tendon it is often impossible to get the heel down in good weight-bearing position because of contracture of the posterior and medial portion of the talo-tibial and talo-calcaneal capsules. Through an incision it is possible to expose these contracted structures so that they can be readily divided. It is surprising to find that many times they are thickened as a result of contracture to about three-sixteenths of an inch. Following disappointing results from posterior capsulotomy alone, it was decided to extend the scope of operation to include the medial capsules. The operation was suggested by those of Ober and Elmslie but is much less radical and therefore less objectionable.

The technic may be briefly outlined as follows: A curved incision is made down to the deep fascia on the inner side of the ankle passing just below the medial malleolus, extending anteriorly to about the region of the navicular bone. It is also carried proximally along the medial side of the Achilles tendon to a point above the ankle joint. The heel cord is exposed and lengthened by one of the numerous methods. The plantaris tendon is usually found to be tight and this is cut. On dividing the deltoid ligament just below the medial malleolus the posterior tibial tendon

is exposed and just under this is the long flexor tendon of the toes. When these are found to be contracted they are lengthened or divided in their sheaths. The medial capsules of the talo-tibial, talo-calcaneal and talo-navicular joints, are exposed when the tendons, vessels and nerves below the medial malleolus are lifted on a retractor and may be readily divided under direct vision. The dissection is carried back to include the posterior capsules. An easy manipulation is sufficient to put the foot in a position of overcorrection and the wound can be closed in layers. A plaster of Paris dressing is applied from the tips of the toes to the middle of the thigh holding the knee straight and the foot in extreme abduction, eversion and dorsiflexion. The circulation of the extremity must be watched carefully because of the marked degree of sudden correction which is obtained. This plaster remains on, usually without dressing of the wound, for approximately six weeks, after which time it is removed so that physiotherapy may be instituted. Further stretching of the foot into overcorrection, baking, massage, and training of the evertor muscles give a good result. The corrected foot should not be allowed to go without the support of modified shoes for weight-bearing and a proper splint for use at night. It is only after prolonged observation that one can be sure that the deformity is finally cured.

In addition to contractures of the Achilles tendon and joint capsules, a tightness develops occasionally in the plantar fascia resulting in a hollow foot deformity. This is usually associated with equinus and sometimes varus and should be corrected before the heel-cord is lengthened. Simple subcutaneous division of the plantar fascia with a tenotome may suffice or it may be necessary to strip off all of the soft parts from the antero-inferior surface of the calcaneus to relieve the deformity (Steindler). In either case a plaster is applied for a month before further corrective work is done.

Transplantation of the peroneal tendons from their normal position under the lateral malleolus to a point anterior to it has been described and by straightening the line of pull of these muscles increased power is supposed to be applied directly to the outer side of the foot. The slack in the transplanted tendons is taken up by the muscles which now, by virtue of their straightened line of pull, have increased power as a corrective factor. The results in our hands have not been satisfactory.

The only bony deformity considered under this heading is an internal torsion in the shaft of the tibia causing the child to toe in. The gait is awkward as a result and weight-bearing in this position favors recurrence of varus of the foot. A transverse osteotomy of the tibial shaft makes it possible to correct the deformity by externally rotating the distal fragment.

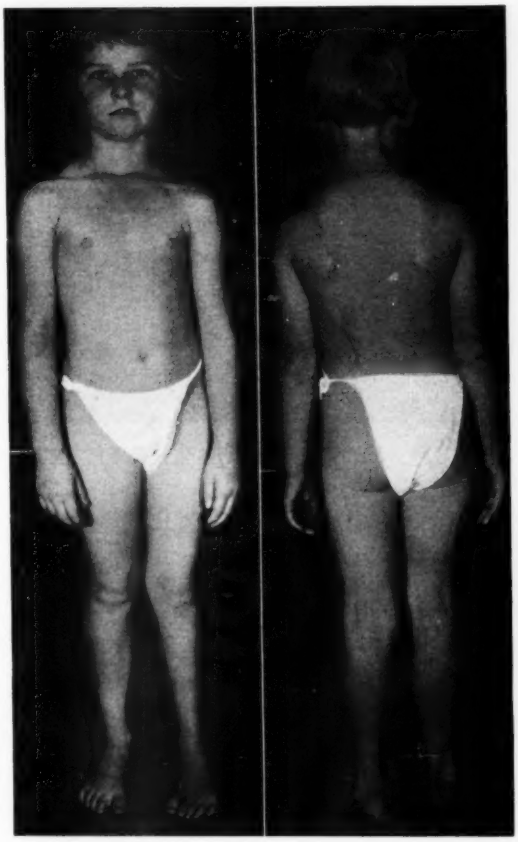

Fig. 3. This illustration shows a common residual deformity following conservative treatment over an extensive period. The inversion of the foot and particularly that of the heel is particularly noticeable.

Treatment of Neglected and Adult Cases.—These two classes of individuals should be sympathized with on two accounts. Firstly, because they have been allowed to grow to adult life with an easily correctable deformity due to ignorance or neglect on the part of their parents or advisors, and secondly, because their deformities

are largely osseous and, although correctable, function cannot be completely restored.

Awkwardness of gait, knock-knees, back and leg aches due to faulty body mechanics, limita-

Fig. 4. The abduction and eversion of the foot and heel has been obtained by a postero-medial capsulotomy by the method described.

tion of activities to the point of complete seclusion in addition to suffering from the painful callosities and trophic ulcers which form on the weight-bearing surfaces are the experience of the adult club-footed cripple.

Much can be done to improve both appearance and function of the deformed feet by surgical means. Before any treatment is undertaken it is highly essential that a radiographic examination be made. The true nature of the deformity can be appreciated when the osseous changes are visualized.

Before any radical procedures are undertaken a maximum degree of correction of the deformity due to soft part contracture should be obtained by forcible manipulation of the feet under anesthesia with the assistance of a Thomas wrench or other suitable lever. Our preference is for the Thomas wrench as it has been very satisfactory in our hands. In addition to wrenching, the contracted plantar fascia and Achilles tendon may be cut. After this more conservative treatment a plaster of Paris dressing is applied holding the foot in the corrected position for a month or six weeks. Further relaxation may take place in the plaster so that the residual deformity is minimized. In the adult the preliminary manipulation and tenotomy can usually be dispensed with.

Complete correction may be obtained by cuneiform osteotomies of the tarsus. The adduction of the front of the foot is corrected by a vertical wedge removed from the outer border in the region of the calcaneo-cuboid and talo-navicular joints, the base being directed laterally. The inversion and to some extent the equinus of the entire foot are corrected by a similar wedge in a horizontal plane. Sufficient bone must be removed to correct or even overcorrect the deformity. After five or six weeks in plaster, bony union is obtained, the malposition of the foot is corrected, and further treatment is unnecessary. Although the flexibility of the tarsus has been destroyed to a large extent, the function and appearance of the deformed foot have been markedly improved.

In the most severe cases and particularly in adults with a unilateral club-foot, the question of amputation of the deformed extremity should be seriously considered. The relief from pain, improvement in appearance, the steadying of the gait and increased ease in walking, and the removal of the consciousness of deformity fully repay the individual for the loss of his extremity. It is, of course, only to be considered as a last resort in the treatment of the deformity.

A brief résumé of three cases will illustrate some of the points which have been mentioned.

Case 1.—A boy four and one-half years of age reported at the Shriners' Hospital in January, 1933. He had a club-foot deformity of a moderately severe degree involving the left foot. Inversion of the entire foot plus plantar flexion and adduction of the forefoot was very apparent (Fig. 1). His treatment had not been started until he was one year of age and then consisted merely of a brace and modified shoe. A very slight degree of correction had been obtained before his admission. Under a general anesthetic his deformity was entirely cured by a subcutaneous division of the plantar fascia and flexor tendons of the toes, forcible wrenching of the foot, followed by the application of a plaster of Paris dressing for six weeks. He was discharged cured of his deformity, wearing a modified shoe for weight-bearing and a splint for use at night. He will be kept under careful observation for a period of two or three years to guard against any recurrence of his deformity.

Case 2.—This patient is a boy of eight years who has a bilateral congenital club-foot deformity of a very severe degree. He had not had treatment of any kind and shows very well the typical picture of neglect. At the time of his admission to the hospital in 1928 he was suffering from pain in both feet, instability of gait

and to a large extent was prevented from living the active life of a child of his age on account of the disability caused by his deformity. A marked inversion, internal rotation and adduction of both feet was very apparent. A large bursa or secondary heel was present on the lateral side of each foot in the region of the

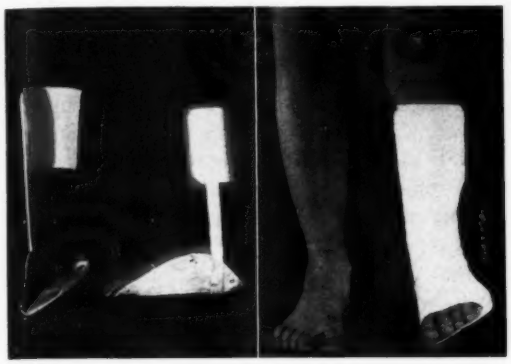

Fig. 5. A simple splint consisting of sheet metal cuff and foot plate connected by a bar on the inner side of the leg is used to hold the corrected club-foot in good position while the patient is off his feet. This splint is bound on with an Ace bandage.

metatarsal cuboid joints (Fig. 2). Following the principle of obtaining maximum correction by conservative means he underwent ten months of active treatment consisting of repeated manipulations under anesthesia followed in each case by a period of rest in plaster and later a subcutaneous division of the plantar fascia was performed, resulting in a fairly good correction of his deformity. At the time of his discharge from the hospital late in 1928 it was felt that he probably would have a recurrence and would need further surgical treatment. In June, 1930, he was readmitted to the hospital and at this time a posterior capsulotomy of the ankle joints was done, combined with the removal of wedges of bone from the outer side of the tarsus as described by Cook. Plaster dressings were applied and left on for six weeks and when these were removed a satisfactory correction had been obtained. Function of the ankle joint was apparently normal although there was limitation of motion in the feet, particularly in the region of the midtarsal joints. There has not been a recurrence of the deformity.

Case 3.—This patient is a girl of seven years who has had treatment since the time she was a baby, with, apparently, a fairly satisfactory correction of the club-foot deformity of the left foot. At the time of admission, however, there was a definite inversion and adduction of the foot, particularly noticeable in the region of the heel (Fig. 3). This was extremely resistant to all manipulation and it was decided to do a postero-medical capsulotomy of the type referred to in the body of the paper. This was done and the correction obtained was very satisfactory. Particular attention is drawn to the fact that the heel is now everted and the adduction of the foot is corrected (Fig. 4). Function has become practically normal due to the very excellent recovery of power in the peroneal muscle group.

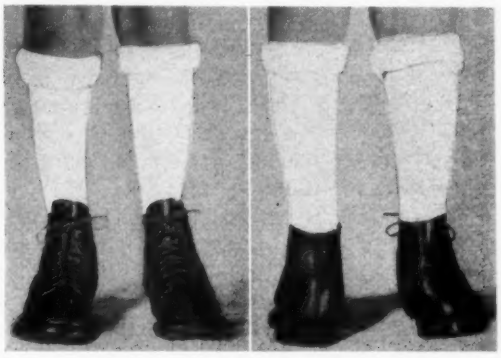

Fig. 6. The modification of the shoes which has been referred to consists of raising the outer edge of the heel and sole ¼ of an inch with a leather wedge and extending the outer side of the shoe laterally about 3/16 of an inch with a metal reinforcement of steel or Monel.

SUMMARY

1. Early gentle and persistent treatment in congenital club-feet yields good results in about 95 per cent of cases.

2. Surgical interference is only to be considered after conservative treatment over nine months to a year has failed to correct or cure the deformity.

3. Forcible manipulation under anesthesia is the first stage of radical treatment and is sufficient in many cases.

4. Tenotomy and capsulotomy are necessary in relapsed or neglected club-feet.

5. Removal of wedges of bone from the tarsus is the treatment followed in neglected and adult cases.

6. Postero-medial capsulotomy for the relief of uncorrected or relapsed inversion and equinus of the heel is described.

7. Night splints and modified shoes as referred to in the text are illustrated.

AMEBIASIS: REPORT OF CASE COMPLICATED BY LIVER, LUNG, AND BRAIN ABSCESS*

J. N. Gehlen, M.D.
Saint Paul

IN PRESENTING this subject† it is desired to emphasize the fact that amebiasis is not only a tropical or subtropical disease, but may be found frequently in our own latitude. Further, carriers are much more common than cases with symptoms of dysentery.

In Minnesota, from 1917 to October 30, 1933, 545 cases of amebiasis with thirty-two deaths were reported. This includes 479 cases, mostly carriers, reported by the Veterans' Hospitals in exservice men. In addition, from 1926 to 1933, The Mayo Clinic reported 132 cases in persons from other states and foreign countries. The writer has histories in his possession of seven cases of amebiasis which occurred in Saint Paul during the past three years, in three of which there were liver abscesses, while the record shows that no cases have been reported to the health authorities from Saint Paul during this period.

Craig[4] believes that about 10 per cent of the population in the United States are infected with *Endameba histolytica*. Williamson et al[12] reported the examination of 148 persons in Chicago in 1929, all food handlers, in whom he found twenty-seven carriers and two persons with symptoms attributable to amebic infection. Andrews and Paulson,[1] in 1930, examined 522 dispensary patients in Baltimore and found only 0.2 per cent infected. Kilpatrick[8] has pointed out that infestation with *E. histolytica* causes dysentery symptoms in an amazingly small percentage of those infected. If we learn to look upon amebiasis as a chronic infection which may affect almost any organ of the body, similar to malaria or syphilis, we shall be better able to diagnose this disease more frequently.

The active, or vegetative, form of the organism dies very rapidly after leaving the body, and is non-infectious, but the encysted parasite may survive for weeks outside of the body at moderately cool temperatures in the presence of moisture. This affords opportunity for spread of infection through water and milk supplies. Infected food handlers with unclean habits may readily pass on the infection to the consumer and constitute as great a menace as a typhoid carrier.

The incubation period varies from several days to several months. The symptoms vary so widely that they may be discussed under three heads as follows:

1. *Carrier or Latent Type.*—There may be no symptoms or the symptoms may be so protean in character that it may be impossible to at once determine whether or not the symptoms are due to the infection. An infected person may complain merely of lassitude, of tiring easily, or continuous headache, or he may present a neuropsychiatric condition. Others may have indefinite abdominal discomfort, slight intestinal disturbance with mild diarrhea and at times constipation. Such vague symptoms may continue for months. Without diagnosis and treatment, liver abscess or other complications may arise.

2. *Acute Type.*—The onset is usually sudden, may begin with headache, nausea, vomiting, chills and dysentery with frequent evacuations (fifteen to thirty a day) mixed with blood and mucus, and colicky pains and tenesmus. In some cases the acute symptoms may develop in a carrier. Fever, though infrequent, may appear. Exhaustion, feeble heart action, cardiac failure and death may follow, or the condition may improve and the chronic form ensue.

3. *Chronic Type.*—The course of the disease may be more or less chronic with attacks of alternating constipation and diarrhea. The stools contain much mucus and sometimes blood. There may be some fever, abdominal pain and tenesmus. There is often progressive loss of weight. The patient may become anemic and possibly jaundiced, and the muscles flabby. Anorexia and flatulence are common. The abdominal wall may be retracted, rigid and tender. The course is variable, not self-limited, and may lead to an incorrect diagnosis of nonspecific ulcerative coli-

*Read before the Staff of Miller Hospital, Saint Paul, November 7, 1933, and also before the Ramsey County Medical Society, Saint Paul, December 6, 1933.

†Since preparing this paper numerous articles on this subject have appeared in recent numbers of the *Journal of the American Medical Association*, one of which has covered the subject of treatment. For this reason the discussion of treatment has been omitted.

tis, mucous colitis, appendicitis or malignancy. If the proper treatment is instituted, some patients will recover entirely and the amebæ in the stool will disappear within a short time. Occasionally the active forms and often the cystic forms persist after all symptoms have disappeared. Complete recovery is often doubtful, for, after an apparent cure, a relapse may occur. Repeated relapses may occur until some grave complication arises. Craig[5] states that probably only one-third of treated cases recover entirely with continued absence of the organisms.

The ultimate diagnosis in amebiasis depends entirely upon the accuracy and persistence in examination of the feces for this protozoa, but first of all the physician must be alert to the possibility of this disease. One negative stool examination means no more than one negative sputum examination in a suspected case of tuberculosis. In acute, fulminating cases when a double infection, amebic and bacillary, is suspected, the subcutaneous injection of one-half grain emetin twice a day for a day or two may be given, followed by a saline cathartic. This may cause the patient to pass a stool mixed with blood and mucus in which the ameba may be found. A sigmoidoscopic examination may give valuable information.

Complications.—Hepatic abscess, single or multiple, is the most frequent complication and may occur at any time, often after all symptoms of dysentery have ceased and sometimes before any intestinal symptoms have developed. The amebæ reach the liver either through the portal vein or possibly by migration through the intestinal wall with direct invasion of the liver. In addition to amebæ, the abscesses often contain a variety of bacteria. In seventy-four cases reported by Craig,[3] the incidence of liver abscess was 33 per cent; in ninety-five cases reported by Harris[6] only 15 per cent. Of 639 cases of amebic liver abscess collected by Rouis,[10] 70.8 per cent were in the right lobe. In 562 cases of liver abscess, Zancarol[13] found a single abscess in 60 per cent.

Rogers[9] recognized a presuppurative stage of amebic hepatitis in which the amebæ lodge in the portal capillaries of the liver. Rapid, painful enlargement of the liver may occur. Rogers believes that the disease in this stage may often be cured by emetin and a liver abscess prevented. The differential diagnosis between the presuppurative and suppurative stage is difficult.

Liver abscess is easily overlooked, as it may develop insidiously and the liver may not be enlarged or tender. A perforation may be the first indication of its presence. Examination may

Fig. 1. Roentgenogram of the lung at the time of admission to hospital. Note a small area of pneumonia and pleurisy, right base.

show an enlarged liver and pain may be elicited on sudden pressure; crepitation at the right base of the lung with dry cough may suggest the condition. X-ray examination may show diminution of the movement in the right diaphragm or the diaphragm may be high and nearly motionless. The leukocyte count may be normal, but when the usual secondary infection is present there is a leukocytosis of from 12,000 to 40,000, with an increase in the polymorphonuclears.

The abscess may perforate spontaneously into the pleural cavity, lower lobe of the right lung, pericardium, or to peritoneal cavity.

Abscess of the lung may be due to direct extension from the liver by perforation through the diaphragm and pleura, or a primary abscess of the lung may occur independently of hepatic disease by the invasion of the ameba from the hepatic veins. Rogers[9] reported lung abscess in 20 per cent of his liver abscess cases.

Brain abscess, according to Kartulis,[7] occurred in 3 per cent of his liver abscess cases. It usually complicates either liver or lung abscess, although two cases are reported in which at autopsy there were no apparent lesions in either the liver or lung. Brain abscess is usually single, but may be multiple.

CASE REPORT

The patient‡ was a local fur dealer, forty-five years of age and married. His family history was unimportant.

In 1917 and each succeeding year he went to Europe, visiting France, England, Germany, Holland, and Switzerland. He had had no illness until after his first European trip, when he developed frequent headaches, thought to be due to sinusitis and for which he consulted numerous rhinologists without relief. In July, 1932, he suffered from an attack of "influenza" and in the same month, while still not well, went to Europe. His headaches became more frequent and he became increasingly irritable. Friends thought the condition was due to business worries, but this he denied. Constipation and occasional indefinite abdominal pain were present, thought to be due to duodenal ulcer. In March, 1933, his headaches became more frequent and intense for which he frequently used 50 to 60 grains of aspirin daily without relief. About April 15, 1933, he became tired, listless and drowsy. This condition increased until April 25, when, while on a trip to Winnipeg, he suffered a slight chill with nausea and vomiting, and complained the next few days of feeling cold.

The patient was first seen on April 22, 1933, when he complained of intense headache and generalized aching. He did not appear sick, was well nourished and mentally alert. The skin had a yellowish tint while the sclera was bluish white. His temperature was 100° F. A few fine, crepitant râles were present at the base of the right lung, posteriorly. The abdomen was slightly distended and doughy in consistency. Neurological examination was negative.

On May 3 he had rigors lasting several hours. His temperature was then 102° F.; pulse 104; respiration 28. Dullness and bronchial breathing were present over the right lower lung area. He was admitted to the hospital when an x-ray indicated irregular consolidation in right lower lobe near the base, with a fuzzy outline of the diaphragm on the right side. Respiratory distress was greatly relieved by the oxygen tent. X-rays of the nasal sinuses were negative. His hemoglobin was 63%; erythrocytes, 4,060,000; leukocytes, 23,360; p.m.n., 94%; lymphocytes, 5%; monocytes, 1%.

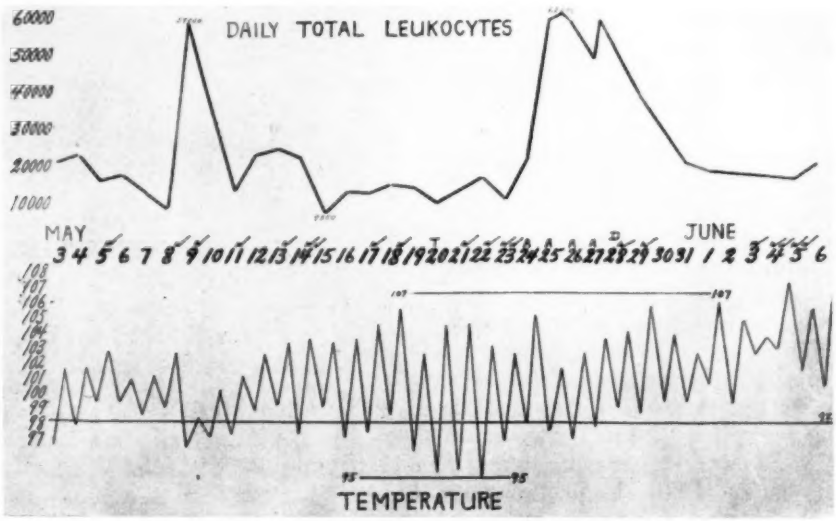

Fig. 2. Daily leukocyte and temperature curve. Chills occurred on dates checked. *A*, aspirations of chest. *D*, drainage of liver abscess.

From May 3 to May 9 he suffered occasional chills and his temperature ranged from 100 to 103° F. During this period additional laboratory tests were made. Blood agglutination tests with *Bact. tularense*, *B. abortus*, *B. typhosus* and *B. paratyphosus* and blood cultures were negative. The van den Bergh test showed: immediate reaction, trace; direct delayed, 3 plus; indirect, 2 plus. Blood smears examined for malarial parasites were negative. Blood chemistry findings were within normal limits. Blood streaked sputum, present in small amounts, showed staphylococci and streptococci only. The urine contained bilirubin. Three stool specimens were examined for amebæ and dysentery bacilli with negative results.

On May 9, the patient's temperature, pulse and respiration dropped to normal for twenty-four hours, to be followed by daily chills and elevation of temperature as shown in the accompanying chart (Fig. 2).

A chest x-ray, on May 10, showed small, pneumonic patches at the base of the right lung with exudative pleurisy; also a small pneumonic patch or infarct in the left upper lobe.

‡This patient had visited Chicago three or four times each year. Although he did not reside at a "loop" hotel, he may have taken meals there.

On May 17 the patient had his first liquid stool. Examination by Dr. K. Ikeda showed many actively motile *Endameba histolytica*. On this date the liver was not tender or palpable but the abdomen was definitely distended and x-ray of the liver and abdomen was negative. Emetin hydrochloride, ½ grain by hypo twice daily, and also stovarsol, grains 4 by mouth three times daily, were begun.

May 18 the temperature was 106° F.; pulse 140; respiration 44. The chills were more severe and a slight cough appeared for the first time. Physical findings remained unchanged.

On May 20 an x-ray taken twenty-four hours after the intravenous administration of thorium dioxide (thorotrast) showed the liver and spleen enlarged but no evidence of a liver abscess. This examination was incomplete since twice the amount of thorotrast should have been given but such dosage was considered inadvisable because of the radioactivity of this drug. A blood transfusion was given.

During the following days there were several liquid stools, one bloody, all containing large numbers of amebæ. Chills and sweats increased in severity. The patient, previously alert, directing his business, now became confused and irrational at times. The urine showed traces of albumin, sugar and a few hyaline and granular casts; also bilirubin. Blood examination showed: sugar, 150 mg.; urea-nitrogen, 20.6 mg.; blood calcium, 9 mg.; blood chlorides, 1.41%.

At 1:00 a.m., May 24, the patient awakened with sharp, excruciating pain in the right chest. A few moments later he was in shock, the entire body cyanotic. He improved remarkably under administration of caffeine sodium benzoate, morphine, and oxygen. A single, flat x-ray plate, taken with a portable unit, indicated a diffuse capacity of the entire right lung with displacement of the heart and mediastinum to the left, obliterating entirely the outline of the heart and diaphragm on the right side. By thoracentesis 1,000 c.c. of dirty, yellowish, purulent fluid was removed. The fluid contained a trace of bile pigment, many pus cells, and an occasional Gram-positive coccus but, as might be expected, no amebæ. Chills and sweats continued with increasing weakness and fever. About 1,000 c.c. of yellow, purulent fluid were obtained daily by thoracentesis. Smears showed immotile ameba-like bodies, many pus cells, Gram-postive diplococci and diphtheroids.

On May 27, on inserting the needle in the tenth interspace in the posterior axillary line, 10 c.c. of thick, greenish, purulent material mixed with blood were obtained. The following day, under local anesthesia, portions of the ninth and 10th ribs were rected in the posterior axillary line and a drain inserted into a large, thick-walled, multilocular liver abscess (Drs. H. B. Zimmermann and E. M. Jones). The abscess contained dark, milky, purulent material of offensive odor. Microscopically there were liver cells, ameba-like bodies, branching mycelia, actively motile bacilli, short spirals and many Gram-positive and Gram-negative organisms, including long filaments and fusiform bacilli.

During the next six days the patient was comatose; chills were frequent; there was transitory right-sided facial paralysis while the superficial and deep reflexes were present and equal. The abscess cavity was irrigated daily, large amounts of pus being removed. Death occurred June 7, 1933.

Fig. 3. Amebic abscess of the liver, surrounded by a zone of fibrosis—an opening into the pleura through the diaphragm, on the right.

AUTOPSY REPORT BY DR. K. IKEDA (ABSTRACT)

Chronic empyema, right pleural cavity, which communicates with the liver abscess through the diaphragm.

A partially collapsed right lung, 700 grams, covered with a thick, purulent exudate. A small abscess 2 cm. in diameter at the base, surrounded by a zone of chronic pneumonia, also an infarct 5x3 cm. above the abscess.

The left lung shows an area of well defined, fresh infarct in the midportion of the lower lobe.

The spleen, 300 grams, shows two, small, yellowish, lumpy areas 5 to 8 mm., representing embolic abscess.

The liver, 2,000 grams, contains an irregular abscess, 2 cm. in diameter, along the dome, which is adherent to the diaphragm. The cavity communicates with the pleural cavity and is lined with rough, yellowish green, velvet-like exudate. Around the main abscess are numerous, small, yellowish, cheesy areas of necrosis, from a few mm. to 1 cm. in diameter, all of which are surrounded by a zone of firm, grayish fibrosis.

The duodenum shows a large, irregular ulcer 13 or 14 mm. in diameter and 8 mm. in depth, with a sharp, overhanging edge, surrounded by a zone of deep, reddish, inflammatory reaction. The ulcer contains fresh blood clot.

In the jejunum are several small areas of yellowish mucosal nodules from 5 to 7 mm. in diameter containing a thick, cheesy material.

In the sigmoid are multiple diverticuli. One of them,

particularly, shows a definite inflammatory reaction around the opening and contains thick, glairy mucus and liquid feces. No other lesions are present in the colon.

Fig. 4. Multiple amebic abscesses of the brain. Note a large abscess in the cerebellum, also a rupture into the lateral ventricle.

The brain shows many irregular abscesses, from a few mm. to 2 cm. in diameter, containing a thick, greenish, sticky pus, scattered throughout the substance of the brain, including the cerebellum. Many are found superficially along the cortical surface; others are deep in the white matter. One abscess ruptures into the left lateral ventricle.

Diagnoses: (1) Chronic amebic abscess of the liver with rupture into the right pleural cavity. (2) Multiple embolic amebic abscess of the brain. (3) Embolic amebic abscess of the right lung. (4) Embolic abscess of the spleen and jejunum (possibly amebic). (5) Acute duodenal ulcer, possibly embolic. (6) Infarcts of the lungs. (7) Multiple diverticuli of the sigmoid with a primary amebic diverticulitis. (8) Chronic empyema of the right pleural cavity.

DISCUSSION

With several consultants, called during the first half of May, the writer considered numerous diagnostic possibilities, including tularemia pneumonia, septic thrombosis of the deep abdominal veins with lung infarcts, malignant endocarditis and undulant fever. By the aid of the parasitologist the true nature of the disease was determined to be amebiasis. It is of special interest that this case presented no dysenteric symptoms until after abscess of the liver and lung developed and that constipation was more or less troublesome until late in the course of illness. Could the headaches which were so persistent over a period of fifteen years have been due to amebiasis? Such cases have been reported. It seems improbable that the brain abscess preceded the liver abscess, which latter presumably was present for several months. The blood picture was unusual, the leukocytosis being much higher than that reported in the literature.

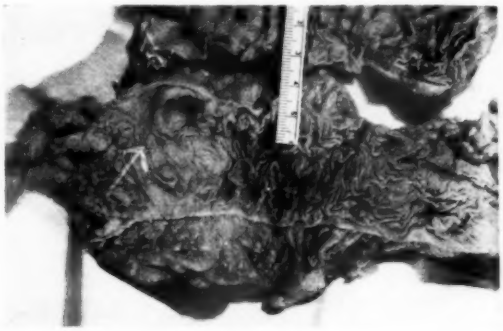

Fig. 5. Primary amebic diverticulitis in the sigmoid.

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TREATMENT OF ENURESIS*

H. S. Lippman, M.D.
Saint Paul

In recent years we have learned to avoid treating symptoms as such. Rather, we have directed our efforts to the uncovering of factors responsible for the symptom, hoping thereby to insure a more permanent result. There is a danger, however, in treating enuresis symptomatically since by so doing we place in one group individuals who are suffering from a variety of conditions. Perhaps a discussion of the treatment will demonstrate this.

Preventive measures demand first consideration. Awakening the child two or three times, if necessary, each night during the second and third years to insure a dry bed will go a long way towards preventing later bed-wetting. The majority of enuretics have not received such training and pediatricians, as a rule, overlook emphasizing the need of such measures, early. The parents must understand the danger of the use of force during the period of training. Calmness and patience with as little display of anger as is possible are short cuts in this training process.

Control of the sphincter is a simple matter in most children. In some, however, despite normal intelligence (and it may be of interest to note that the average enuretic child rates well on intelligence tests) and a real effort on the part of the child to coöperate, training is not established. These failures can be attributed to such factors as differences in bladder and urethral irritability, the reception of impulses in the central nervous system, the ability of the kidneys to concentrate, the temperament of the child, and the temperament of the parents.

Let us assume we are dealing with a boy in whom attempts at training have been unsuccessful. He is ten years old and is sensitive because of his failure to be trained. He has been given many forms of treatment, none of which have resulted in permanent relief. He comes to the physician for treatment and appears to be anxious to coöperate. To begin with, he is given the opportunity to talk the whole matter over with the physician who, through sympathy and understanding, attempts to gain his confidence. The parents are kept strictly out of these interviews and the boy understands early that they will no longer assume the responsibility for the treatment. He is told that he has indicated a wish to get well, that we accept him at his word and are prepared to help him. One cannot overestimate the value of the relationship that can be set up between an unhappy child and an understanding adult who remains undisturbed by failures to respond to treatment. Because of this relationship he may, for the first time, consistently cut down on fluids late in the day, refrain from too active play after dinner, and retire on time. He may make a strong effort to respond to the alarm clock and set it again for the next awakening. Success may follow these simple measures previously attempted without results. The boy is asked to come in once a week with a chart upon which he has indicated whether or not the bed has been dry at different periods during the night. In most instances any child who is coöperative can be assured a dry bed if he is awakened frequently enough during the night. It is often necessary to awaken him quite frequently at the risk of losing needed rest in order to assure him that he can have a dry bed several nights in succession. This gives to the child the encouragement that is necessary to continue with treatment. By degrees the number of awakenings can be lessened and the bed will, in all probability, remain dry.

Unfortunately, every child who professes a wish to get rid of his enuresis does not remain coöperative. This may be due to the fact that he has never learned to do things for himself, or that the symptom is too deeply rooted and he is less anxious to get well than he had imagined. Under such conditions the treatment must be referred back to the parents.

It may be well at this point to emphasize the inadvisability of continuing routine measures over a long period of time. Too often, a child is placed on restricted foods, fluids or exercise, or given a medication long after its inefficacy has

*From the Amherst H. Wilder Child Guidance Clinic. Read before the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, May 22, 1933.

been demonstrated. Atropine, for example, useful in some cases has little value in others, and this should be quickly determined.

The child is told early that the symptom is a stubborn one and that many changes may be necessary before the final treatment will be found. His weekly reports will be honest if he is certain that the physician respects him for his effort.

Day and night urines are routinely examined to rule out infection and to determine his ability to concentrate at night. Fluids are increasingly limited. He is given exercises towards strengthening the sphincter muscle. Recently, we have been using the salty diet recommended by Krasnagorski, which aims to retain fluids in the tissues. We are as yet unable to decide on the effectiveness of this treatment.

If treatment directed towards physical factors is unsuccessful we search for those of an emotional nature. It is unnecessary to explain to anyone who has tried to treat enuresis that emotional factors play an unusually important role. The plan of treatment now changes. Frequently the restrictions above referred to are removed. The awakening at night by the use of the alarm clock, however, is usually continued.

The interviews are directed primarily towards the building up of a feeling of confidence. He is allowed to speak of his parents, his brothers and sisters, teachers, classmates. As he becomes more confiding he has the courage to tell of his jealousies, hatreds and fears. He dislikes, particularly, to tell of his fears. He hates to be considered a coward and yet most enuretics are weighted down by fears and worries. Later on in the interviews when he is no longer ill at ease the subject of sex must be discussed. His anxieties in connection with masturbation and other sexual irregularities must be talked through. He learns that he is no different than a great many others and that there is little danger of insanity or severe illness resulting from masturbation. Through his getting rid of guilty feelings in connection with his practices and fantasies, he is more apt to lose the insecurity that has helped to keep him from getting well. One needn't be a psychiatrist to carry on this treatment. As a matter of fact, the family physician or pediatrician may have the advantage over the psychiatrist through the fact that he is already known and respected by the family.

We must keep in mind that we are dealing with a child who has suffered. If our efforts to help him meet with failure we can let him go with the feeling that he will probably soon outgrow the habit and be like other boys. He should never feel that we are disappointed in him, so that he can still accept himself as an individual of merit.

We have learned a great deal from those who have worked intensively with problem children. It is to be hoped that the work that is at present being done by psychiatrists and psychoanalysts on the problem of enuresis will give us a clearer insight into the emotional conflicts underlying the enuresis. We are very much in need of this insight.

SARCOMA OF SECOND METACARPAL BONE: CASE REPORT AND REVIEW*

ALBERT E. OLSON, M.D.
Duluth

OUR knowledge of bone sarcoma has greatly increased in the last few years. On the other hand, any thorough review will emphasize the fact that there is still considerable confusion on the subject. This confusion is apparent in a study of the etiology, pathology or, for that matter, almost any phase of osteosarcoma. For this reason and in conformity with the literature on the subject I am not hazarding a positive diagnosis but confining myself to an opinion based on a clinical, radiological, and pathological study of this case.

In November, 1929, H. C., a male, twenty-eight years old, a steel worker by trade, received a crushing blow on the back of his right hand. He stated that at the

*From the Arrowhead Clinic, Duluth, Minnesota. Presented at the April, 1933, meeting of the St. Luke's Hospital Staff, Duluth.

time he was aiding in removing a large bolt, with a chisel and sledge hammer. The hammer missed its mark, striking the hand above the knuckle of the right index finger. The back of the hand swelled, became discolored and painful.

The man worked in spite of moderate pain for three months and the swelling increased. A pimple formed and later discharged a watery fluid. He consulted a physician who diagnosed the condition osteomyelitis and advised draining the bone.

On March 3, 1930, the bone was curretted. No pus was found and the attending surgeon stated that the bone did not have the gross appearance of osteomyelitis. Repacking and further treatment continued for months, the wound failing to heal and the swelling of the hand increasing. Red tissue filled the center of the rapidly growing ulcer.

This patient came under our observation in October, 1931. A radiograph of the hand (Fig. 1), taken at this time, revealed a swelling of soft tissue and bone destruction. Dr. Gage Clement, radiologist, was called to examine the hand and advised the use of preoperative x-ray therapy because of the probability of malignancy. A specimen (Fig. 2) was removed for analysis. The second and third metacarpals and corresponding fingers were almost sloughed off so that removal was easy. There was a fracture of the second metacarpal. A view of the back of the hand, after removal of specimen, is also of interest (Fig. 3). Dr. Berdez, pathologist, reported spindle cell sarcoma and his report of this specimen is as follows:

"*Gross.*—This is a specimen weighing 255 gms. The specimen includes the index, the third finger of the right hand, together with the corresponding metacarpal bones. The soft parts of the anterior and posterior surfaces of the hand corresponding to the bones removed are included in the specimen. The specimen measures from the tip of the third finger to the proximal extremity of the specimen, 17.2 cm. in length. It measurers 6.5 cm. in width and up to 7.4 cm. in thickness in the region corresponding to the distal half of the metacarpal bone where the hand is extremely thickened. The skin of the palmar surface of the specimen shows an irregularly ulcerated area, measuring 2.2 cm. in diameter. In the center of the area there is an opening of a fistulous tract which leads toward the middle part of the second metacarpal bone. On the radial side of the specimen, the skin shows a large area of ulceration measuring 4x4.5 cm. The edges of the ulcerated area are quite irregular and lined with skin which is quite ulcerated in places. There is apparently little tendency toward healing. In this ulcerated area there are two other irregular openings leading also toward the second metacarpal bone. The walls of the fistulous tracts are lined by some edematous tissue of granulation. On cut surface the mass which is protuding on the palmar surface of the hand is formed by a whitish, compact tumor tissue showing in places a few small, cloudy, yellowish specks. The tumor involves also the second metacarpal bone *which is fractured just beyond its middle.* The tumor apparently also extends to the third metacarpal bone. The musculature of the palmar surface of the hand is infiltrated by the tumor masses. The interosseous muscles between the second and third metacarpal bones are also infiltrated.

"*Microscopic.*—The tumor tissue is formed by numerous, mostly spindle shaped, cells which are sometimes arranged in bundles. The tumor cells (Fig. 4 to 10, inclusive) show oval or round vesicular nuclei. A moderate number of mitoses are present. In places the tumor tissue shows fibrillar elements, at other places the tumor tissue shows small areas of necrosis. At the

Fig. 1. Radiograph of hand taken in October, 1931.

Fig. 2. Specimen removed for analysis.

Fig. 3. Back of hand after removal of specimen.

periphery, the tumor tissue shows a tendency to infiltrate the surrounding tissues.

"*Diagnosis.*—Sarcoma of the hand."

The specimen was sent to Dr. Bell of the University of Minnesota for corroboration. His report was:

"The tumor from the hand of H. C., sent to me by Dr. Berdez, shows a sarcoma. The histologic type is largely spindle cell. I do not see any bony tissue in the substance of the tumor but it may have arisen from the bones of the metacarpus. The prognosis is very bad but an occasional case survives. There is no treatment but amputation."

Amputation below the head of the humerus with complete dissection and excision of lymphatics of the axilla was performed and the patient made an uneventful operative recovery (Fig. 11).

A radiograph taken on February 5, 1932 (Fig. 12), before operation, showed an area in the lung which we considered metastatic invasion. Dr. Clements' report on the latter was as follows:

"The right lung shows a small spherical area of increased density, 2 cm. in diameter, just outside and below the hilum in the parenchyma of the lung which is, undoubtedly, a metastatic new growth. The left hilum is also exaggerated and there is some interstitial fibrosis over both lung fields."

A recent radiograph (Fig. 13), taken April 25, 1933, shows recession of the involved area and no new areas have been visualized. The patient has not coughed and has retained his normal weight. He has received the usual postoperative roentgen therapy with apparent beneficial effect.

This case is of interest because it illustrates

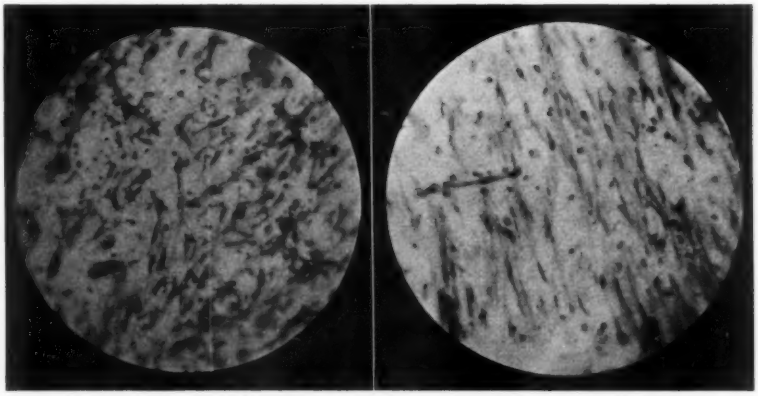

Fig. 4 *(left)*. Proliferating fibroblasts which show no definite arrangement.
Fig. 5 *(right)*. Fibroblasts show beginning parallelism. One capillary seen apparently as yet without lumen.

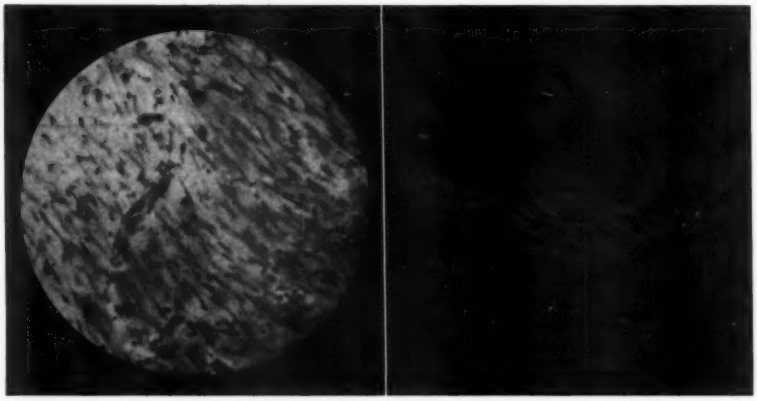

Fig. 6 *(left)*. Zone of transition from the fibroblasts to the tumor tissue. The lighter half of the picture is formed by fibroblasts; the darker and more cellular part is tumor tissue: the tumor tissue is infiltrated with a few lymphocytic cells. A better capillary is also present. The tumor tissue consists here of cells resulting from proliferation of connective tissue cells.
Fig. 7 *(right)*. Here the tumor cells are more densely grouped into intertwining bundles.

many of the problems arising in a study of osteosarcoma. The rarity of the tumor in this location, the association of trauma with subsequent tumor development, the neglect of tissue examination at first operation, the uncertainty of the origin of the tumor and the question of amputation in the presence of pulmonary metastases, warrant further consideration.

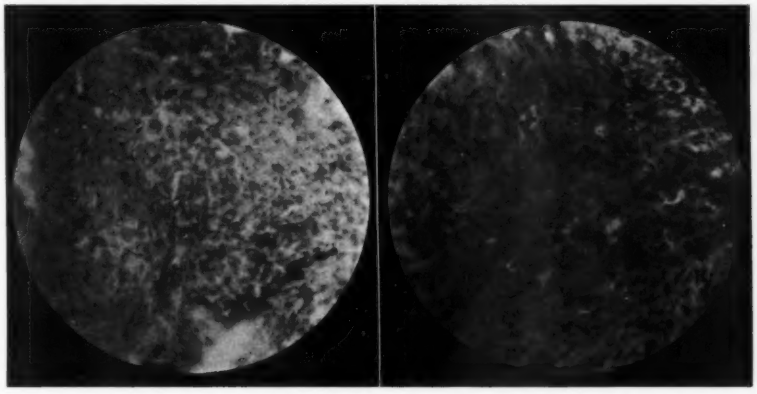

Fig. 8 *(left)*. A small area of tumor tissue in which the tumor cells, instead of being spindle shaped, show a tendency to become polyhedral or round.
Fig. 9 *(right)*. A similar area where the tumor cells show the same tendency to a more marked degree.

"The incidence of primary malignant bone tumors is best seen from the fact that about one case out of three of sarcoma of the human body is a "bone sarcoma" (Kolodny[11]). These tumors arise from cells which are supposed to form the most firm and stable tissues and yet osteogenic sarcomas rank with the most malignant new growths.

The femur, tibia and humerus are most frequently involved. This being a metacarpal sarcoma, it is interesting to know that metatarsal and metacarpal sarcomas are extremely rare (Coley[4]). Ewing[7] quotes Christiansen's table showing that sarcoma of the bones of the hand occur in less than 2 per cent of cases.

It is significant that "osteogenic and giant cell tumors occur mainly at the ends of the long bones where the growth period is longest, the natural momentum of growth greatest and exposure to trauma most likely" (Ewing[7]). The ends of the diaphysis are most often the seat of osteogenic tumors; the ephiphysis, of giant cell tumors, and the shaft of myelomas.

The association of trauma cannot be denied in many cases. Statistics vary. Coley[4] found such history in 40 per cent in a series of 300 cases, Gross[9] in 50 per cent, Meyerding[14] in over 50 per cent, so that one can safely assume that from 40 to 60 per cent present histories of trauma. I think almost everyone will agree that it is not difficult to elicit a history of injury in almost all cases settled under compensation laws during the present depression. Local injury to cells must follow trauma and it is quite possible that atypical division and growth can develop. Necrosis, hemorrhage, aberrant growth in the granulation tissue may be disturbing factors.

Certain rules have been formulated in determining whether a given trauma is an exciting factor in etiology. Haas[10] enumerated four as follows:

1. Definite proof of sufficient trauma at site of tumor.
2. No prior gross lesion or other injury or disease in same region.
3. Sufficient time elapsed from time of injury to allow for development of tumor.
4. Definite signs of tissue disturbances should persist from the time of injury to the time of development of the tumor.

R. Baumann-Wattwil[1] believes that there is a definite relation between trauma and subsequent tumor development. He enumerates four rules for consideration:

1. Given accident must be single.
2. Lesion produced must be considerable.
3. There must be some relation between site of accident and tumor.

4. Time relation between occurrence of trauma and appearance of tumor must be reasonable, namely, 3 weeks to 2 years.

A most important and obvious conclusion regarding the relation of injury to tumor formation is stated by Hass,[10] namely: "All of the traumatic explanations fail without assuming a previous abnormality, because most injuries are not followed by sarcoma."

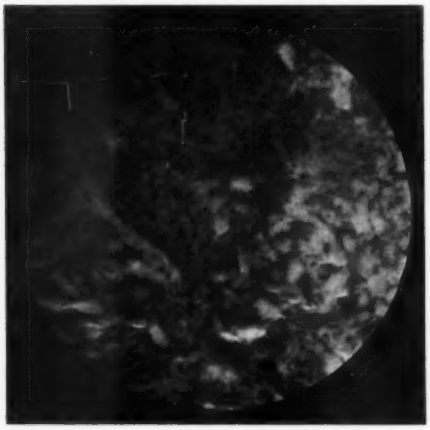

Fig. 10. An area of tumor tissue at the edge of which there is a marked tendency for the fibrillar elements to become hyaline. The tumor cells between the hyaline elements are small, atrophic.

One cannot pass the subject of trauma without stressing the necessity for removal of tissue in all cases demanding operation. The tissue removed may not contain the malignant involvement, but the effort is worthwhile. Formerly, dissemination was feared, but this probably occurs only in exceptional cases. It is well to study all cases of fracture, especially the ones that follow rather insignificant traumata. A pathological fracture following primary union of a fracture after open reduction is illustrated in Lewis' *Practice of Surgery* (pp. 29, 30). The original specimen removed was reported as consisting of granulation tissue. Following operation a large sarcoma developed. A subsequent study of the original specimen showed the presence of sarcoma at the time of open reduction. This illustrates the value of removing tissue in bone operations for microscopic examination in cases of doubtful etiology. This case would have been recorded as sarcoma following fracture if the original specimen had not been saved and thus shows the difficulty of histologic diagnosis.

Kolodny's[11] classification of bone sarcoma is as follows:

1. Osteogenic sarcoma.
2. Ewing's sarcoma.

Fig. 11. Patient following operation. Uneventful recovery.

3. Myeloma.
4. Unclassified sarcomas, including the extraperiosteal sarcoma and the angio-endothelioma.
5. Giant-cell tumour (benign).

Copeland[6] divides all osteosarcomas into two large groups, namely: (a) those related to osteogenesis; (b) those of non-osseous origin.

Inasmuch as the tumor in the case reported probably originated from bone, we shall consider here only the first group. Osteogenic sarcoma is a comparatively recent name given to those tumors derived from mature bone cells or from cells retaining a potential ability to form bone. It does *not* mean, necessarily, that the tumor thus formed must contain bone, production of bone being merely one of the properties of the cells.

The relationship between bone development and tumor formation is not generally appreciated. It is commonly assumed that a permanent completely developed skeletal structure is achieved early in adult life. Copeland[6] states that the "development of the human skeleton is never complete and that at all ages there are transitional forms persisting between component parts of bone."

Osteogenic sarcomas are of mesoblastic origin and as such may exhibit any of the characteristics of cells from the primary undifferentiated

spindle cell to the mature differentiated osteoblast. Osteogenic sarcomas originate from the osteoblast at some stage of its development: fibroblast; chondroblast; osteoblast.

Early and persistent pain at the end of one of the long bones is one of the first symptoms (Coley[5] and Ryerson[15]). Benign tumors rarely cause pain. Osteomyelitis usually causes fever and leucocytosis. Palpation may aggravate the pain. Pathological fracture may occur when the tumor develops sufficiently, as it did in this case. General dissemination occurs in almost every case within the first three years. It was formerly thought that the blood stream was the only means of transmission. It is now known that the lymphatic system occasionally transports the tumor cells. Pulmonary metastases are most frequent. Articular cartilage is very resistant to tumor invasion so that joint invasion is rare.

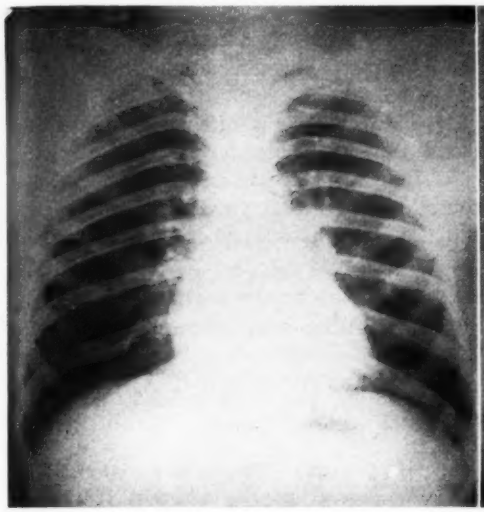

Fig. 12. Radiograph taken February 5, 1932, before operation. Note area considered metastatic invasion.

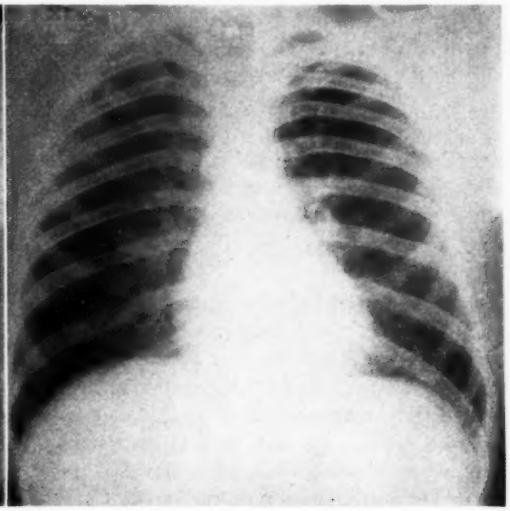

Fig. 13. Radiograph taken April 25, 1933, showing recession of involved area.

The diagnosis of bone sarcoma thus becomes doubtful in the primary stage. Given a spindle cell sarcoma one cannot definitely state that it is a fixed fibroblast—capable of no further development than fibrous tissue or that it is osteogenic, undifferentiated and potentially able to develop into the fixed osteoblast and develop bone.

It is, therefore, obvious that a pathological diagnosis alone may be insufficient in determining whether the origin of a sarcoma is from bone or surrounding connective tissue. A section of the bone itself would be valuable. Desjardins states that, in many quarters, the diagnosis of the pathologist is accepted too blindly and often clinical, radiologic, and other factors are not given due consideration. The opposite is often true and some of Coley's work discredited because of omission of pathologic proof. The diagnosis must rest on all three angles and even then, sometimes, definite statements cannot be made. Regressive changes occur after radiation and inflammatory changes sometimes confuse the picture. The difficulty in differentiating some highly cellular granulation tissue from sarcoma is well known.

Treatment may be classified as follows:

1. Surgery
   - (a) local excision
   - (b) amputation
2. Roentgen and Radium Therapy
   - (a) preoperative
   - (b) postoperative
   - (c) palliative
3. Coley's Serum

As in most malignancies these cases are usually seen too late for proper treatment to be given. Usually a combination of the treatments mentioned is used. Preoperative and postoperative radiation to the operative field and any metastases may be given. Magnusson[12] advises against

the use of radiotherapy alone, while Coley[5] advises early amputation in osteogenic sarcoma followed by toxin treatment. This method, in comparison with preliminary radiation and subsequent amputation, produced better results. Prolongation of life in irradiated cases is usually noted and pain is often relieved. Whether amputation should be done in the presence of metastases is doubtful. Gibbon[8] reports a case of a five-year cure following amputation with metastases present, the latter being treated by x-ray. Behring[3] believes that biopsy aggravates the condition, but whether performed by punch method or open method, Coley[5] believes the advantages far outweigh the disadvantages.

The proper use of Coley's serum demands considerable study. It is a combination of the toxins of streptococci and bacillus prodigiosus and its use based upon the fact that patients having had erysipelas rarely develop malignancy and that if the disease develops during malignancy, regression occasionally has been noted. It is often used in combination with radium by Coley[5].

The prognosis in osteogenic sarcoma is poor in the majority of cases. Most of the patients do not show the marked cachexia noted in carcinoma. In 200 cases of osteogenic sarcoma accepted by the Registry for Bone Sarcoma of the A. C. S., only thirteen five-year "cures" were noted, and in nine of the thirteen, the histological structure was sufficiently atypical to raise doubt on the part of some of the reviewing pathologists as to the true nature of these tumors. Gibbon[8] states that five-year cures are of the rarest occurrence. Magnusson[12] (Stockholm), reviewing thirty-nine cases from 1910 to 1928, recorded only seven alive, and Behring[3] (Lund) an average postoperative survival in 384 cases of about one year. Gross[9] (Vienna, 1870) measured a rapidly growing spindle cell sarcoma and found that it grew 33 inches in thirty-four months. Others are more slow growing. Adolescents are more prone to rapid invasion and metastatic involvement than patients over thirty years. In young children, growth is even more rapid. The longer it takes the tumor to develop, the more unlikely it is to be extremely malignant. As to location in relation to the degree of malignancy, medullary and periosteal types show the same mortality. Brophy's grading should be fully utilized in determining prognosis.

SUMMARY

1. Osteogenic sarcoma occurs most frequently during the second and third decades.

2. It is frequently associated with trauma.

3. Microscopic examination of all removed tissue is essential.

4. The cell type is often not fully differentiated, thus confusing the pathological picture.

5. Pulmonary metastases are not always a contraindication to amputation.

6. The prognosis is poor, but an occasional patient survives.

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THE ALLERGIC FACTOR IN MIGRAINE

H. B. Sweetser, Jr., M.D.
Minneapolis

IN discussing migraine, we may well start by discussing what we mean by migraine. In this study I have taken it to be a symptom complex consisting of a prodrome which may be a night of unusually good sleep, an unusual feeling of well-being, or a vague ill-defined depression. The prodrome is often absent. In a larger number of patients there is an aura, varying in different patients but almost always the same for the individual. This may be a feeling of numbness, or tingling anywhere in the body, usually the face, a one-sided blindness, flashes of light before the eyes, and other sensations. After this has been present from five minutes to several hours the headache comes on. Neither the prodrome nor the aura is necessary to the attack, and quite often the headache is present on awakening in the morning. The headache is usually one-sided, but may change from side to side during succeeding atacks and indeed during one attack. The ache is usually severe and may be actually prostrating. A personality change may occur which disappears at the end of the attack. Nausea is almost invariably present and usually vomiting. Occasionally the vomiting is the signal of the end of the attack. After the headache has lasted from twelve to forty-eight hours, it slowly disappears to be followed by a sequel which is again a remarkably recurrent sensation in those who have one. The most common one is a sense of complete fatigue, but it may be a dizziness, a craving for acid or unusual food, or the sufferer may feel better than in days. After the attack there follows a refractory period during which there is no recurrence no matter what is done. Not many patients with migraine have all the symptoms described, but all have headache and almost all have one or more symptoms.

The simplest description of allergy that I have found has been one by Rich in a recent paper on "Allergy in Tuberculosis":

"When a foreign protein finds its way parenterally into the animal body, certain changes take place in the body as a result of contact with their protein. The exact nature of these changes is still obscure, but the effects which they may produce if the same protein reaches the tissues again, are quite familiar in the guise of the various 'hypersensitive reactions' to foreign protein. Depending upon conditions the reaction may appear in the form of anaphylactic shock, asthma, hay fever, serum sickness, food allergy, the Arthus phenomenon, or the tuberculin reaction. While these various conditions appear at first sight to be very different from each other, there is persuasive evidence that underlying all of them is the fundamental circumstance that the exposure of the body to a bland undigested protein, stimulates the production of a specific antibody, different for each protein, which has the power of reacting with the protein in some way, and that the different forms of hypersensitive reaction are results of the interaction of antibody and protein antigen under different conditions."

To connect these two apparently entirely unrelated conditions at first sight seems difficult. However, if the underlying mechanism of migraine is considered as follows, its allergic character may appear quite natural. From a study of the literature and a series of patients, it seems to me that the ultimate lesion may well be a wheel or a localized angioneurotic edema in some brain area, which may vary in location in different attacks but which disappears without trace. A transient edema is one of the characteristic evidences of an allergic reaction, *i.e.*, the result of interaction of antibodies and a protein antigen.

From a clinical point of view, the evidence in support of this proposition is: (1) the attacks are periodic and followed by refractory periods; (2) some cases follow Mendelian laws while others occur sporadically in allergic families; (3) age incidence corresponds to that of other allergies, with the onset most frequently in the first decade with a gradual decline in incidence toward middle life and few occurring after the fourth decade; (4) predisposing factors appear to operate as in other allergies; (5) eosinophilia occurs during the attacks; (6) many patients give positive skin reactions; (7) patients with migraine have more than their share of other allergic diseases.

From this point of view I have studied twenty-

*Read before the Hennepin County Medical Society, Minneapolis, March 15, 1933.

three patients. Of these seventeen had a history of migraine among parents, brothers and sisters, twenty-eight such relatives in all. Other allergic conditions were present in the families of eleven. Among the patients themselves, eighteen had other allergic conditions such as asthma, hay fever, urticaria, canker sores, eczema, Raynaud's disease, and susceptibility to plant poisons. The incidence of canker sores was especially high and in a remarkable number their occurrence was diminished by elimination diet.

In studying these patients, all were thoroughly interviewed not only as to their medical history, but also regarding food habits and special dislikes and idiosyncrasies. In addition to careful physical examinations skin sensitization tests were performed on almost all, using groups of antigens as arranged by Dr. Ellis of the Students' Health Service at the University of Minnesota.

In treatment, exclusive attention was paid at first to diet, eliminating all foods giving positive skin reactions and those to which the patient had a special dislike or a known idiosyncrasy. Rowe's elimination diets were also used. In the few patients who failed to get relief, other methods of treatment were also used later.

Of the twenty-three patients treated, seventeen gave their diets a fair trial. Of these, eleven obtained marked relief; three were somewhat improved, and three were definitely unimproved. Six we were unable to follow. The outstanding feature in the result of treatment was that the closer the supervision, the more intelligent the patient, and the closer the diet was followed, the better were the results.

CASE REPORTS

One young woman of twenty-nine had had severe headaches since the age of twenty-one. They occurred every four to fourteen days, and were severe enough to put her to bed for a day or two in almost every attack. By eliminating just eggs she had been entirely relieved of these attacks for over two and a half years. In that time she has had two attacks, one caused by eating an egg intentionally, and another by eating egg whites unknowingly in marshmallow candy. Thinking of a psychic factor we had her take an pseudo eggnog and she had no attack whatever. She also has slight attacks of nausea without headache after eating nuts or raw apples.

A woman of forty-four had had attacks of migraine since the age of thirteen. Each attack began with a "sinking in" of her right eye and she would "lick the teeth" on the right side. Then she would have a pain in the right side of her head lasting twenty-four hours. She also suffered from severe hay fever. Elimination of coffee, fish, beets, spinach and onions, relieved her almost entirely, though she likes her food and eats of the "forbidden fruit" occasionally with reappearance of her headache. This woman had been treated with calcium and many other remedies previous to diet control.

A woman of thirty suffered from attacks of migraine since the age of three. Elimination of eggs, chicken, shellfish and walnuts has rendered her free of headaches since June, 1932, whereas previously they had recurred every three to seven days. This woman and both parents are quite brilliant but very erratic. Her two sisters, father, and maternal grandmother had migraine, and her mother suffers from hives after eating strawberries and tomatoes.

A woman of thirty-three, suffering from headaches every seven to ten days since before the age of ten had positive skin reactions to onions, cabbage, banana, and chocolate. She has seven brothers, four sisters and a father who have migraine. For thirteen months she has had attacks only when she has broken her diet.

A woman of thirty-two who had been having family trouble had attacks of sick headache since the age of fifteen. They began with what had been called spastic colon, which, after continuing for a day, gave place to a sick headache which was always heralded by pain in the angle of her right eye. She had found, herself, that these attacks followed the eating of fish and chocolate especially, and also of onions, melons, and bananas.

Since 1924, when Vaughan reported four cases of migraine relieved by diet elimination, there have been a number of additional reports, the outstanding ones being a series of sixty-two cases by DeGowin of Michigan, brought on by food and relieved by elimination of the same food; of 202 cases by Balyeat with over 80 per cent relief from food elimination, and by Rowe of California who reported relief in about 78 per cent of cases. In my small series the percentage of almost complete relief was 62, but many of my patients were seen at the Dispensary and I feel that even better results could have been obtained with closer supervision and better cooperation on the part of the patients.

In conclusion, I believe it is established that there is an allergic factor in the majority of cases of migraine and that treatment based on this conception and the use of diet control will effect a large percentage of good results. This percentage varies with the persistence and skill of the physician and the persistence and cooperation of the patient.

THE "DO'S AND DON'TS" OF THE INJECTION TREATMENT OF VARICOSE VEINS*

H. O. McPheeters, M.D., F.A.C.S.
Minneapolis

THE writer has so often been asked what to do and what not to do in the injection treatment of varicose veins that he has felt that a paper bearing directly on this phase of the subject would be very timely. With that idea in mind this article is presented on the "Do's and Dont's" of the injection treatment of varicose veins and ulcers.

Each case should be studied separately as to the history of its onset and progress. When did the veins in this particular case develop? Are they associated with injury, infection, pregnancy, phlebitis or are they congenital? Different types of veins will demand different treatment and precautions should be taken in some of them. If the patient gives a history of phlebitis, when did the phlebitis develop? Was it postoperative, postpartum, associated with pneumonia and was it of the superficial or deep veins? Was it localized or extensive? Does the history indicate that there was extensive damage done to both the venous and lymphatic systems. Was that damage more or less transient? Was it only in one leg or was it bilateral?

If there are ulcers present when did the first ulceration occur and what was its relation to the development of the varicose veins? Is the ulcer present a true varicose ulcer?

Other factors which should be taken into consideration are the age, habits and occupation of the patient. Judge and expect results according to the age of the patient and the circulation of the extremity. Sedentary habits and occupations are not conducive to rapid improvement of varicose ulcerations. On the contrary the patient should be instructed to walk a great deal, as will be discussed later.

PHYSICAL EXAMINATION

Much in the preceding paragraphs can only be gained during the course of a combined history taking and physical examination. It is impossible to elicit a complete history from the patient in the consulting room and it is only during the course of the examination that you are able to fully and completely arrive at the facts in the history of the case.

A routine general physical examination including that of the urine should be done on every patient. Neither a high nor low blood pressure is a contra-indication per se.

If the patient has diabetes and has bad varicose veins he should be treated, but naturally sugar preparations should be avoided. Be careful of your technic and avoid sloughs. If the patient has nephritis avoid any solution that might be an irritant to the kidneys.

If the patient has a cardiac condition with large varices present he or she will get wonderful relief and improvement in his or her general condition following a cure of the varicose veins. It is important, however, that no cases of broken compensation should have their veins treated during the decompensated state. *Never* treat any patient that is bedfast for any reason, cardiac or otherwise.

Definite pelvic pathology may or may not be a factor in the development of varicose veins. I believe that the patient has less possibility of post-operative complications if the veins are treated first and the pelvic operation put off until a later date.

The presence of "Resting Infections" so thoroughly discussed by deTakats[2] and others,[6] should be recognized. It is far safer to treat this before the injections are started. It is best treated by local heat, moist and dry, with the patient in bed for a few days. The leg should be supported with bandages, stocking or casts when the patient is up and about.

Some of our most gratifying results have been obtained in the treatment of varicose veins during pregnancy. Here invalid mothers at mid-term have been made comparatively comfortable throughout the rest of the term.

Due recognition of the endocrine factor in the individual case cannot be emphasized too strongly. The large plethoric, hypothyroid individual

*Presented before the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, May 24, 1933.

with ulcerations on the lower legs is an example of this type of a case. Here we find large saccular veins feeding the ulcerations and lying in the superficial fat, which has become to a great extent fibrosed as a result of the chronic infection present. In addition to the sluggish circulation of the extremity, the patients are often so obese that they cannot get about with sufficient agility to properly make use of the effect of the calf muscles in walking. Experience has shown that the veins do not thrombose so well when embedded in thick fat while recurrences and new formations occur much more frequently. An evaluation of the above factors should be made during the examination.

ACTUAL TREATMENT OF CASE

a. In the injection treatment of varicose veins the one most outstanding, important and emphatic "Do" is that the patient *must* be kept ambulatory. The one great advantage of the injection treatment over the operative treatment is that the patient may continue to be about his work. It is imperative that the patient be ambulatory because the circulation of the leg is markedly increased by the systolic and diastolic action of the calf muscles when the patient is walking. When the patient is ambulant there is a very positive back pressure and reverse flow in every case of fully developed varicose veins. This backward pressure tends to force the thrombus more firmly down into the varicose vein and tends to prevent the formation of an embolus.[9] This is exactly contrary to the idea held relative to this work previous to the past few years. Some surgeons in the past have insisted on their patients having bed rest for a period of ten days following treatment. Not so any more.

b. The factor of speed in the treatment of the individual case varies a great deal. In some it is of no importance whatever. Some patients would prefer to keep coming to the office twice a week for several months instead of having the work done quickly; other patients are on a vacation and wish to have work done then, while others come from a great distance, at much expense, and it is very important that the work be completed in the shortest time possible.

c. The temperament of patients must be considered. Some patients are of the lethargic hypothyroid type which do not notice pain or distress to speak of. That patient can be treated completely at one sitting and not mind it. If she has been subject to much pain and distress during her life she may not even notice or complain of the soreness occasioned by the chemical phlebitis following the treatment. On the contrary there are many patients of the nervous, neurotic type, under high tension, who have never been subject to pain or distress and who complain markedly about the mere prick of the hypodermic. Often a patient will faint while being given a single injection. In such a case make no attempt to give extensive treatment at a single sitting but instead advise the patient to have this treatment completed in many sittings with one or two injections at a time. Let the extent of the treatment at a single sitting be in a measure judged by the size, extent and location of the veins. As stated above, never treat the patient so extensively, completely and radically that he will be incapacitated and made bedfast. The veins about the ankle seem to be the most tender and painful of all, following injections. The veins in the popliteal space also give the patient much distress and if extensive or large and saccular should be treated separately from the rest in order that you may carefully continue the post-operative support to the chemical phlebitis or venitis which develops.

d. Finally, treat every patient according to the theory of the treatment. The theoretical principle underlying the injection treatment of varicose veins is that we wish to inject into the vein lumen some substance which will react on the endothelial cells of the intima, causing a definite injury to it which will provoke the formation of a thrombus. These solutions are prepared in many different strengths and with varying destructive powers. It must be remembered that stagnation of the blood current in the vein and localization of the injected fluid are the two principal factors in this treatment. Next, the solution that we inject should be injected in a known strength. By that I mean that if we wish to use a 5 per cent, 50 per cent, or 75 per cent solution we should do our best to inject that particular strength and then retain it so. Do not inject any solution into a vein full and distended with blood, because of the dilution which will result. This dilution, of course, cannot be accurately measured. With experience one can determine the preferable strength to use, as well as the length of time that it should be held localized in order to obtain results. This will naturally vary with the

solutions used and with the types of veins in different locations. The varicose veins of blond individuals seem to be more easily affected than those of dark brunettes. The venitis is more severe in blonds. Finally, in order to accomplish the above theoretical principles each doctor must use his own judgment as to the method which can most definitely accomplish the desired results.

MY OWN IDEAS

a. The theory of the treatment is, as stated above, that we should inject into the empty vein some destructive solution of a known concentration, retaining it there under control as long as is necessary. This is done by making use of tourniquets and the back pressure from the deep veins outward through the communicating veins. This is easily controlled by either elevating or lowering the extremity being treated. Of course it is impossible to introduce the needle into an empty vein but theoretical perfection should be approached as nearly as possible by any method preferred. The work done and presented elsewhere[9] by the injection of skiodan under the fluoroscope proves most clearly that the solution can actually be localized and retained where desired by the above method.

b. The blood pressure readings[9] prove the source of the reverse flow and why the tourniquets are of value.

c. Endeavor to inject the right amount of solution. Never inject more than enough to barely fill the varix being treated. It is wrong to inject large amounts of any solution into any vein filled or distended because that solution will not be retained locally, but will pass directly through the communicating veins into the deep system. In small amounts this would cause no damage, but if large amounts are used and the patient's circulation is poor and tourniquets are applied real tightly there is definite danger of producing an injury to the deep veins.

d. The number of injections at a sitting vary with the individual case. Often I give only one injection, while in other cases I have given as many as twenty-five to thirty.

e. At each sitting when the treatment is finished, it is of *definite* value to drain the injected solution from the treated area, otherwise there is a possibility that it may continue with its destructive action locally longer than desired and may cause true complications such as sloughs or extensive periphlebitis. When the treatment is finished, elevate the leg, drain the solution out, withdraw the needles and apply gauze sponge pressure directly over the needle puncture before the extremity is lowered.

f. The postoperative care and treatment of these cases is most important. In all this work I wish to emphasize either *do it right or leave it alone*. I am more positive all the time of the relative value of the minor points in the treatment and these are in no place more outstanding than in the post-operative care. You first do your best to retain the solution locally just long enough to give you the desired destructive result. Then attempt to collapse the large varices so that when the resultant thrombus forms it will be not more than one quarter inch in diameter instead of as large as your thumb. The venitis and periphlebitis following the treatment causes pain and tenderness and the support of the Ace bandage, adhesive plaster, Elastoplast bandage, Unna's cast or the elastic stocking should be given. The choice will rest with the physician and the case. Local heat, either moist or dry, also tends to relieve the soreness of the reaction.

g. In order to obtain the best results the doctor must carefully and consistently give follow-up care and inject all new varices appearing or reinject those veins not completely and perfectly thrombosed. Lack of attention to these points explain most failures.[1, 4, 5, 8]

h. I am convinced that it is most essential to have the great saphenous completely and firmly thrombosed to the groin in order to assure a permanent result. It is difficult to locate this at times except by means of the P.P.T. (percussion pulse transmitted).[8] By this simple percussion test the great saphenous is very easily located although it may lie deep in the fat and not be detectable by ordinary means. It is useless to expect a permanent result following the injection of veins in the lower leg, regardless of what solution is used, as long as we have the hydraulic effect of the great saphenous, open as large as your thumb, with a reverse flow forcing downward from above tending to recanalize the treated and thrombosed veins.

SOLUTIONS USED

The choice of solution varies with the individual physician as well as the patient to be treated. There are many solutions that are very

effective from the clinical viewpoint and results obtained. I have discarded practically all of the solutions causing intense and severe cramps because many patients are reluctant to return for further treatments on account of them. Solutions causing the most severe cramps are: (1) sodium salicylate; (2) sodium chloride (20 per cent); (3) the sugar and salt mixtures. The latter, however, are very efficient when used with discretion and the associated use of a moderate sedative. For the majority of my work I prefer, first, sodium morrhuate in the 3, 5 and 10 per cent strengths; second, the quinine and urethane solutions, and third, the sugar and salt mixtures.

COMPLICATIONS OF TREATMENT

a. The most common complication following the injection treatment is the development of a phlebitis either chemical or infectious. The infectious phlebitis should not occur but at times it happens even with the best of technic. deTakats has discussed this at length under the subject of "Resting Infections."[2, 6] The best treatment is bed rest, hot packs and elevation of the affected part.

b. The occurrence of sloughs should be a thing of the past. If they do occur, treat them exactly the same as a varicose ulcer. The application of hot packs for the first twenty-four hours very materially lessens the pain. Following this, apply the rubber sponge and Ace bandage just the same as to an open ulcer. When the slough and gangrene shows its line of demarcation then excise it as it separates. Do not attempt to completely excise the slough early and hope that it will heal by first intention, as it seldom will do so. The open ulcer which will remain when the wound breaks down and lies wide open following the attempted excision leaves much more of a scar after its healing than would the original slough.

c. I do not believe that gangrene of a leg can develop following the injection treatment of varicose veins as the solution is injected into the vein and not into the artery. Such cases have been reported. Gangrene of an extremity can come only from a blockage of the arterial supply and I do not see how it would be physiologically possible for this to develop following the injection of any solution into a vein.

THE COMPLICATIONS AND END-RESULTS IF VARICOSE VEINS ARE NOT TREATED

a. The most common complication is the development of an acute, infectious thrombophlebitis. This may be either of the superficial or deep system, and it may be either localized or extensive. For all cases of infectious thrombophlebitis of the deep system I believe the most satisfactory treatment is rest in bed with the extremity in elevation and wrapped in hot packs. The application of the electric baker for half an hour twice a day seems to be an adjunct to this treatment. If the phlebitis involves the superficial veins all the way to the groin then the same treatment is used. *If* the phlebitis is *localized* to an area in the *superficial* system with large loops of veins above open and filled with blood but not thrombosed, the treatment is entirely different. Here the author would emphasize the immediate injection and thrombosing of the veins above the area.[6] This I believe is a very marked advance in the treatment. In the past it has been absolutely forbidden to inject any vein for a period of one to three years following the presence of an infectious thrombophlebitis. We know now that this is positively wrong. For this kind of a case use quinine and urethane or sodium morrhuate. Do not inject one segment and then leave the rest but on successive days inject those segments above until the vein is completely thrombosed to the sapheno-femoral opening. In the presence of a resting infection or acute phlebitis in a great saphenous vein which is unusually large, I advise high ligation as close to the femoral opening as possible.[3, 12] Start the injections the day after the ligation. Do not forget the use and value of external support to all these inflamed areas during the course of treatment after the patient is up and about.

b. Another complication is eczema and pruritis. Do not hesitate to inject the veins associated with this condition. The veins are the causative factors and the condition will improve more rapidly following their injection than it will following the application of solutions and ointments alone. The combined use of both methods is by far the best.

c. Varicose ulcers are a very common and serious complication. Make a careful differential diagnosis of the case as the first point in the treatment.

TREATMENT OF VARICOSE ULCERS

If varicose veins are present and associated with varicose ulcers then all that has been said above applies.[11] In fact, it is more important and urgent to inject the varicose veins here than in the simple uncomplicated case. There usually is much congestion, edema and cellulitis associated with the ulceration. There is no object in injecting a vein in an area of cellulitis but the vein above and outside this area of cellulitis should be injected as soon as possible. The technic of the injection in such a case is just the same as discussed above. Clinical experience has proved that veins will thrombose very poorly in areas of extensive cellulitis, edema and fat. These cases should be treated by injecting the veins above, with the application of the ulcer treatment to the ulcer and cellulitis area below.

Too much importance cannot be given to the actual treatment of the ulcer itself with its associated area of cellulitis and edema.[7] This has been discussed fully elsewhere and I only wish to reëmphasize that treatment. The mechanics of the rubber sponge and Ace bandage in connection with the systolic and diastolic effect of the calf muscles while the patient is walking is clear and positive. Clinical experience most perfectly bears out the accuracy of the theory underlying the principle. Dress the ulcer with pure, thick balsam of Peru. Over this area place a gauze dressing with a layer of some mild ointment, then apply a sufficient amount of gauze and sheet wadding to protect the skin from the rubber sponge which is then applied accurately over the ulcer. Be sure to apply a good grade of rubber sponge and not a soft one. The more firm and resilient it is the better. This is bound in place with a plain gauze bandage. The entire lower leg including the rubber sponge is then wrapped tightly with a four-inch Ace bandage. This entire dressing should be changed every two to four days.

In all extensive cases of varicose ulcers use the seed implantation graft of Braün.[11] Continue the use of the rubber sponge and Ace bandage following the skin graft the same as for any ulcer. Many ulcers seem to heal and close rapidly following adhesive strapping, restrapping ulcer every five to seven days. Following the healing of the ulcer be sure to continue your supportive treatment. The Unna's cast is best for the first month or so followed by the elastic stocking or bandage for several months thereafter. Support should be maintained to the extremity as long as any edema is present.

The plethoric hypothyroid individuals with ulcerations need endocrine medication and reducing diet as well. They will recover more rapidly and the veins will thrombose more firmly when injected if they are put on a strict obesity diet with thyroid medication until they show a definite *plus* basal reading. The addition of parathyroid to the treatment very definitely helps some patients, though not all.

In conclusion let me list the "Do's and Don'ts" of the injection treatment as follows:

"DO'S" OF THE INJECTION TREATMENT

1. Study the case history.
2. Examine case carefully.
3. Watch for areas of "resting infection."
4. Keep patient ambulatory.
5. Consider temperament of patient.
6. Choose solution for the case.
7. Follow theory of treatment.
8. Mark veins to be injected while patient is standing.
9. Adjust posture of leg and patient during treatment to suit the individual case.
10. Leave tourniquets applied five to ten minutes after injections.
11. Make multiple injections if needed.
12. Apply and maintain gauze sponge pressure at point of injection immediately upon withdrawal of needle.
13. Inject all patients that show a positive, negative or double Trendelenburg. Use Perthe test in doubtful cases.
14. Inject pregnant women the same as others. Give more care to post-injection support in these cases.
15. Inject labioceles when painful.
16. Inject patent varicose veins starting from four to six inches above area of acute infectious thrombophlebitis and continuing to groin.
17. Re-inject vein if thrombus is not hard up to saphcno-femoral opening.
18. Ligate pre- or postoperative as preferred. Ligate high or not at all.
19. Anticipate and explain soreness following inpection treatment.
20. Support inflamed veins.
21. Inject all varicose veins in ulcer cases as soon as the edema and cellulitis have improved.
22. Use rubber sponge and Ace bandage on all ulcers and sloughs.
23. Wrap ulcer and sponge tightly. Emphasize walking.
24. Use Braün seed skin grafts for all large ulcers.
25. Use rubber sponge pressure and support following the implantation of skin grafts.
26. Strap ulcer with adhesive if it does not respond to the usual sponge treatment.

27. Continue support and care to ulcer cases after they are healed.
28. Remember the association of a hypoactive thyroid and parathyroid with ulcer complications.
29. Study and treat each and every case individually.

DON'TS OF THE INJECTION TREATMENT

1. Don't overtreat patient. Keep him ambulatory.
2. Don't worry about emboli but remember their possibility.
3. Don't forget asepsis. Always be careful.
4. Never inject if not sure needle point is in vein lumen.
5. Don't inject if you have punctured posterior wall of vein.
6. Don't be afraid to inject veins in thigh up to groin.
7. Don't inject large amounts of any solution in one varix.
8. Don't inject if vein is distended.
9. Don't inject over crest of tibia or over malleolus.
10. Don't forget that a quarter inch thrombus is the ideal thickness.
11. Don't forget that all extensive cases of varicose veins show a Trendelenburg positive or double test.
12. Don't excise sloughs. Use hot packs early. Later treat same as ulcers. Remove necrotic tissue.
13. Don't use silver nitrate for one week after the implantation of the seed grafts.
14. Don't hesitate to give patient a few more injections than you planned at first without extra charge.
15. Don't claim or promise too much. Recurrences and new formations do occur.
16. Don't discharge patient. Watch for new veins every six months to one year.

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CASE REPORTS

FECAL INCONTINENCE WITH MEGACOLON*

T. L. Birnberg, M.D.
Saint Paul

We are presenting the following case of chronic fecal incontinence in a child because of: (1) its rarity; (2) its definite association with a spina bifida; (3) the fascinating study of neurological function that it offered, and (4) the therapeutic problem it presented.

The patient was a boy seven years of age, who was first seen August 26, 1932.

The family and past history were irrelevant.

His complaint was a fecal incontinence of six months duration. Prior to that time he had no incontinence at any time, nor has he ever had any eneuresis.

Since the onset, the child would have incontinent stools two to three times a day. He would pass small stools—usually hard, marble sized balls, but at times he would have a mass of softer, but definitely normal, pasty feces in the gluteal fold. This incontinence was more frequent in the evening but might occur at any time of the day or night. The patient seemed to pass these small stools without any conscious recognition of the act and the small hard lumps were frequently found scattered about the house. The child sometimes appeared ashamed of his incontinence; at other times he seemed in no wise disturbed. However, he is not a mentally retarded child and is up in his school work.

Physical examination was negative throughout, his height and weight being about normal.

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Digital examination of the rectum showed the external sphincter possibly a little relaxed. There was a constant large fecal mass in the rectum. The sphincter reflex was normal and there were no sensory or motor disturbances of the gluteal region or of the lower extremities.

Chirurgical for 1930, report a case in a girl of twelve years who had a fecal incontinence from infancy. The only abnormal physical finding was a relaxed, gaping anal sphincter. X-ray showed a spina bifida of the second sacral arch. Freeing the nerves from the mass of fibro-adipose tissue by operation resulted in a cure.

Fig. 1 Fig. 2 Fig. 3 Fig. 4

Fig. 1. Barium enema showing marked dilatation.
Fig. 2. Partial evacuation of enema showing diminution of size and return of haustrations.
Fig. 3. Installation of air following evacuation of barium.
Fig. 4. High barium enema five months later: showing that although patient has been functionally well for five months, the bowel still dilates when overloaded by barium enema under pressure.

Sigmoidoscopic visualization showed a widely dilated rectum and sigmoid. There was no evidence of mechanical obstruction (Dr. H. W. Christianson).

Opaque enema showed a very large, redundant colon *with a constant mass of feces in the upper rectum.* Following partial evacuation, the colon was less large and showed fairly good haustrations. No obstruction of any kind was observed.

X-ray examination of the spine shows a lack of fusion of the first sacral arch, appearing as a spina bifida occulta. The spinous process of this segment seems entirely absent.

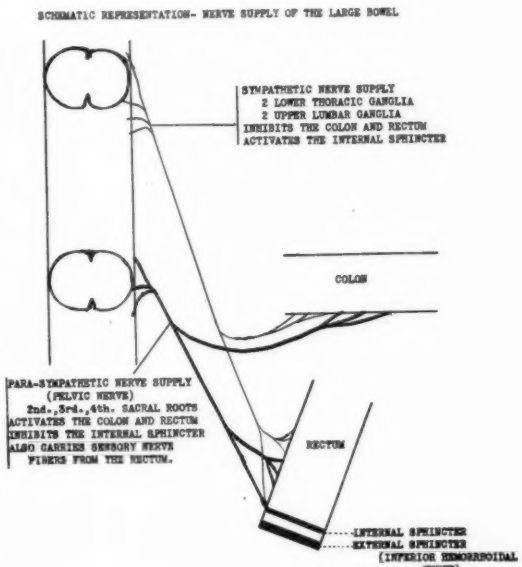
Diagnosis: (1) Fecal incontinence; (2) Megacolon; (probably secondary to a spina bifida occulta).

In so brief a time the differential criteria can be only briefly mentioned.

Could this megacolon be congenital, that is, the Hirschprung's type? Hirschprung's disease is usually associated with an obstinate constipation from *early infancy*—sometimes accompanied by periods of paradoxical diarrhea; further a large abdomen is always present. This patient gave no such history, the first disturbance being noted at six years. Megacolon secondary to peripheral obstruction is frequent in adults and rare in children but is well ruled out here by x-ray and proctoscopic examinations. We feel that this moderate megacolon is secondary to the spina bifida.

In spina bifida occulta the bony defect and the associated dural defect are filled with a mass or pad of lipomatous and fibrous tissue, which usually extends down into the spinal column and may envelop some of the spinal nerves. Impingement upon nerves occurs from contraction of the fibrous tissue bands or from the relative shortening of the spinal cord that results from the less rapid growth of the cord as compared to the vertebral column. The resultant symptoms depend on the nerves involved, and vary from urinary incontinence to flaccid paralysis of the lower extremites.

In a rather thorough perusal of the literature we frequently noted the statement that spina bifida might be a cause of fecal incontinence, but definite case reports seem lacking. P. Santi et Cibert in the Lyon



These men make no statement of the neuro-mechanism involved, but the findings suggest involvement of the spinal nerves to the external sphincter.

Brief mention may be made of the physiology of the lower bowel from a neurological standpoint, in the light of our present knowledge.

The true sympathetic group of nerves, formed chiefly by the ninth thoracic to the fourth lumbar ganglia with their corresponding rami communicantes, maintains a functional unit, and may be termed the "filling nerve,"

since it has an inhibitory action on the large bowel and is the activator nerve to the internal sphincter.

The parasympathetic group, formed by the second, third, and fourth sacral ganglia with corresponding communicating rami to and from the cord, makes up the pelvic nerve, which may be termed the "emptying nerve" since it activates or contracts the colon and rectum but inhibits or dilates the sphincter. Further, this nerve group carries the sensory fibers from the rectum—a point of importance in this case.

It is the balanced interaction of these mechanisms that controls bowel function and reflex defecation. That involvement of this mechanism may produce megacolon has been shown experimentally by Adamson and Aird (British Journal of Surgery, October, 1932). These men produced a progressive megacolon in cats by the surgical section of the parasympathetic roots, thus causing a loss of parasympathetic activation of the large bowel with a resultant sympathetic inhibitory preponderance.

Since a spina bifida at the first sacral arch might involve any of the communicating rami of the parasympathetic ganglia, and particularly might involve the afferent fibers from the rectum which are concerned not only with reflex mechanisms but also with cerebral consciousness of rectal filling and emptying, the following deductions as correlated to the presented case seem probable:

1. Loss of parasympathetic activation of the large bowel resulted in a partial megacolon, noted only if the colon was overloaded, and not associated with muscle hypertrophy or primary colon disease.
2. The reflex act of defecation was interfered with.
3. The loss of filling sensation in the rectum suggests how small amounts of stool might have been passed without the knowledge of the child.
4. Loss of sphincter tone occurred because of: (a) the persistent presence of a large fecal mass in the rectum, mechanically producing a partial paralysis of the sphincter; (b) lack of stimulation to the sphincter through the reflex arc tonus.
5. Frequent small stools were the result largely of a mechanical process. The small hard lumps were forced out by the accumulating fecal mass in the dilated rectum. Clinical observation verified this: when the child had a full rectum, incontinence occurred; when his rectum was kept fairly empty, no incontinence occurred.

Treatment was directed at decreasing the normal strain on the weakened functional control of the affected region of the bowel. Primarily it was necessary to keep the rectum fairly empty to avoid overload and to help the sphincter resume its tone. This was done as follows: The child was given frequent enemata to keep his rectum empty. He was given a low residue diet and a large amount of carbohydrate to favor bacterial growth and produce a soft, acid stool. Phenolphthalein was given to stimulate peristalsis. For about three weeks the patient did not show much change; then a rapid improvement occurred. For the past five months he has had absolutely no incontinence. He has usually two soft, acid stools daily. The sphincter tone has improved, indicating that the previous large fecal mass had affected its tonus. The colon and rectum are still enlarged, if overloaded (as by a barium enema), but the size diminishes considerably on partial evacuation, indicating an absence of obstruction or of muscle wall disturbance.

At present the boy not only has no incontinence but is much happier, eats well, and is gaining nicely. Though not cured physiologically, he is apparently cured clinically. The bowel will probably function normally, if the weakened portion is not given an excessive load.

SUMMARY AND CONCLUSIONS

1. A case of spina bifida occulta with resultant megacolon and fecal incontinence is presented.
2. The rarity of spina bifida with fecal incontinence and no urinary incontinence is noted.
3. The neurogenic mechanism of lower bowel control is described.
4. The mechanism of spina bifida in causing fecal incontinence and megacolon is discussed.
5. Medical therapy directed at overcoming the faulty physiology was given and a clinical cure observed thus far for five months has been obtained.
6. The necessity of surgical treatment, with its risk and uncertain results, has been at least temporarily obviated.

PRIMARY CARCINOMA OF THE LIVER*

REPORT OF CASE IN A SIX MONTHS OLD INFANT

GEORGE K. HAGAMAN, M.D.
Saint Paul

This case is being reported as a very rare and interesting condition, and as a reminder that in the differential diagnosis of any enlargement of the liver, malignancy must be considered, regardless of age.

In 12,000 autopsies, White reports only eleven cases of primary carcinoma of the liver but does not mention the age incidence.

Briggs, in a recent résumé of the literature states that Griffeth collected fifty-seven cases of primary carcinoma of the liver in children from one to sixteen years of age, and Danse collected twenty-three cases in children less than two years old.

Carcinoma of the liver is usually an expression of malignant changes in other parts of the body but the literature on primary malignant changes in the liver is being constantly enlarged.

Time does not permit of any enlargement upon etiology, types, or liver changes in these cases.

The patient was a six months old white infant entirely breast fed from birth. Delivery had been normal. There had been no intercurrent illnesses. The family history was entirely negative. There was no history of malignancy in the family.

Nothing unusual was noticed until a few weeks before admittance to the hospital, when the baby became fretful. The stools were greenish and more frequent. One week before a physician was consulted, the mother had noticed the baby's abdomen was becoming larger and he was acting as if in pain. At this time Dr. Beals was consulted. A few days later the abdomen became suddenly greatly distended. I saw the baby with Dr. Beals on March 25th. The baby was extremely pale and the abdomen was greatly distended. The liver was tremendously enlarged and numerous nodules were felt over the surface of the liver.

On admission to the Children's Hospital, the blood showed the hemoglobin to be 34 per cent, white cells 17,750, red cells 2,350,000. The differential count was normal. A liver puncture was done and blood under great pressure was obtained. The child expired on the second day after admittance and an autopsy was granted and performed by Dr. Ikeda, the essential parts of which follow.

NECROPSY FINDINGS

The body is that of a male child 67 cm. in length and approximately twenty pounds in weight. Rigor is absent. No hypostasis, no cyanosis or demonstrable jaundice. The skin and mucous membrans are pale and waxy. A puncture mark is present in the mid-epigastrium over an abdominal mass which occupies the upper half of the abdomen and distends it enormously.

The subcutaneous fat is scanty.

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*Read before the Annual Meeting of the Minnesota State Medical Association, Rochester, Minnesota, May 22, 1933.

The peritoneal cavity contains a few cubic centimeters of fresh blood. The liver occupies two thirds of the cavity. It extends 12 cm. down from the costal margin in the right mid-clavicular line, 10 cm. from the xiphoid process and 4.5 cm. from the left costal margin. In the mid-axillary line, it extends 11 cm. from the costal margin on the right and 2 cm. from the margin on the left side. The right lobe thus extends clear across the abdomen to the left flank. The liver is loosely adherent to the diaphragm, which is thickened, roughened and hemorrhagic on this side. The appendix is normal. The diaphragm extends to the fourth rib on the right and to the fifth rib on the left.

Fig. 1. Photograph showing the marked enlargement of the abdomen.

Fig. 2. Lateral x-ray of the abdomen showing the tumor mass.

Fig. 3. The liver removed and incised.

The liver weighs 1,040 grams. It is 25 cm. across, 14 cm. in height (above downward) and 13 cm. in thickness (before backward). The left lobe shows a smooth, pale, yellowish-brown capsule. The right lobe is rounded and the superior and inferior surfaces show irregular, nodular, subcapsular masses. The capsule is adherent to the diaphragm lightly. The capsule is stretched and shows irregular yellowish and reddish spots of the tumor through it. The cut surface of the right lobe shows a large, well defined, rounded, nodular tumor mass about 13 cm. in its greatest diameter occupying the substance of the liver with a narrow zone of parenchyma partly around it. The mass consists of a soft, mushy, greenish-gray tumor tissue with areas of hemorrhage. The center of the mass is occupied by a large clot of blood and serum from which liquid blood escapes freely. The tumor mass is definitely demarcated from the surrounding parenchyma and the capsule, which is thickened and tightly encapsulates the greater part of the mass. It bulges out on the cut surface and is easily broken. The cut surface of the left lobe is pale yellowish-brown, cloudy and fatty. The gallbladder is normal. The gastrointestinal tract is normal.

The spleen, kidneys, adrenals, testes and thyroid glands are normal.

ANATOMICAL DIAGNOSES

1. Primary adenocarcinoma of the liver with central necrosis and hemorrhage.
2. A single metastatic nodule in the right lung.
3. Probably direct extension of the tumor to the right diaphragm.

Liver.—The tumor consists of medium size cells with hyperchromatic nuclei and a spacious, granular "foamy" cytoplasm with tendency to form cords with arrangement simulating the hepatic architecture. More or less fat globules are noted in the cells, in some areas this feature predominating the picture. Large blood spaces, areas of hemorrhage and necrosis of the tumor tissue are noted in various part of the section. Wide bands of connective tissue extend into the tumor from the thickened capsule. Massive infiltration of leukocytes and proliferation of fibroblasts along the periphery of the blood mass is seen. No bile pigments are noted within or about the tumor cells. No biliary ducts are observed. Section of the left lobe shows cloudy swelling and fatty infiltration of the parenchymal cells with a slight atrophy of the cords.

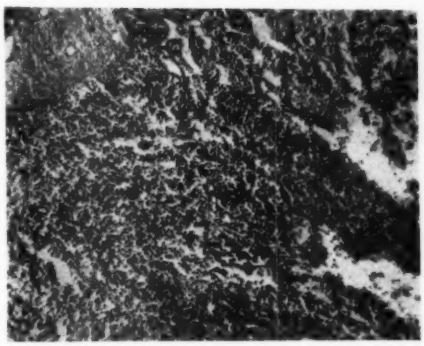

Fig. 4. Microscopic appearance of primary adenocarcinoma of the liver.

Lungs.—Lung shows a single well encapsulated nodule about 5 mm. in diameter consisting of the cells of the type noted in the liver tumor. The cells form a more solid mass but show fine alveolar septa with suggestion of cord arrangement. Several large, blood filled spaces are noted within the tumor. Otherwise, lung is normal.

Diaphragm.—The muscles are definitely hypertrophied. The hepatic aspect is covered with a thick, irregular layer of organizing fibrin-blood over a narrow zone of granulation tissue showing fibroblastic proliferation, hemorrhage, edema and a few infiltrates. Whether or not some of the cellular elements are tumor cells can not be stated definitely.

MICROSCOPIC DIAGNOSES

1. Primary adeno-carcinoma of the liver (liver cell type).
2. Metastatic nodule in the lung.
3. Non-suppurative inflammation of the diaphragm with peri-hepatitis.

DOUBLE ILEAL INTUSSUSCEPTION

J. E. Engstad, M.D.
Grand Forks, North Dakota

J. J., aged 5, was admitted to my services at the Deaconess Hospital, March 3, 1931, giving a history indicating intestinal obstruction. The young patient was in a state of collapse with rapid pulse, subnormal temperature, and marked abdominal distention. A large dense mass could be palpated in the ileocecal region. After a stimulating regime, he was taken to the operating room with the diagnosis of intestinal obstruction.

Under ethyl anesthesia, a McBurney's incision was made and considerable clear fluid escaped through the incision. A large mass was found inside the lumen of the cecum with a dense pedicle in the ileocecal valve. A double ileal intussusception into the cecum was determined.

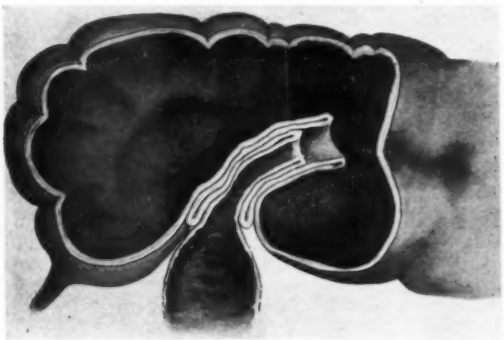

Fig. 1.

With gentle traction the intussuscepted loop was delivered out of the cecum. The ileum was found to be necrotic for a distance of about 6 inches, extending at its distal end to a point a little over a half an inch from the cecum. This small distal end of the ileum was of sufficient length to serve as a good cuff for an end-to-end approximation.

The necrotic mesentery was first excised by a V incision of sufficient depth to include all the thrombosed blood vessels, and all bleeding points were ligated. The gut was then milked empty of its contents and clamps properly applied. The necrotic mass was excised in such a manner that no infective material touched the mesenteric part of the bowel. A modified lock stitch, including both layers, joined the severed ends, leaving as small a hem as possible with a view to minimizing the hazard of a damming action on the intestines. The serosa was further reinforced by a number of Lambert silk sutures. The toilet was most difficult, due to the enormous dilation of the bowels, which had extruded from the abdominal cavity during our rather hurried technic. Some relief was accomplished by aspirating the gas from a couple of the largest loops. The incision was loosely sutured with catgut, a rubber gauze drainage being instituted.

In the middle of the night I was hurriedly called to the hospital and on inspection a large mass of intestines were found under the bandage, the intra-abdominal pressure having forced the intestines through a very small space between the sutures. The patient's general condition had improved, and his bowels were moving freely. All sutures were severed, and all loops replaced with the exception of two or three segments that seriously objected to being put back in their normal position. They were left outside, as the danger of sharp kinking and agglutination was of greater menace than their extrusion.

The bowels were dressed with warm vaseline twice a day, and covered with warm, moist saline gauze. A marked atelectasis of the right lung occurred, which was a factor which precluded any extensive manipulation of the intestines. The extruded loops thrived well, and functioned perfectly. Gradually as the pulmonary complication improved, the segments were from time to time forced back into the cavity, but part of one segment became agglutinated to the edge of the incision, and refused to budge. Gradually a film covered the extruded loop, which in time contracted to such a degree that after six months' time the skin had closed over the wound, the fascia had approximated, and there were no evidences of any firm adhesion of the segment to the serosa.

The patient is enjoying excellent health up to date. On straining, only a slight bulging is demonstrated under the scar.

HEPATEX P. A. F. NOT ACCEPTABLE FOR N. N. R.

The Council on Pharmacy and Chemistry reports that Hepatex P. A. F. (Evans Sons, Lescher & Webb, London, England; Thomas A. Hedley, New York, American agent) was submitted for the Council's consideration as another liver preparation for intravenous and subcutaneous administration in the treatment of pernicious anemia. It is stated that 5 c.c. of the preparation represents 100 Gm. of mammalian liver. The information supplied concerning the preparation of the product appeared to be in most respects consistent with the methods described by Cohn et al. for purifying liver extracts suitable for intramuscular and intravenous administration; however, the statement is made that from the watery extract "the protein matter and other inactive constituents are removed by a special secret process," and no further information as to this process was supplied. The firm was informed that the product might be made acceptable (a) if the proprietary name was replaced by a descriptive name, such as "Liver Extract for Intramuscular Use"; (b) if the recommendation for its intravenous use was discontinued or convincing evidence of its safety submitted; (c) if the objection to the "secret process" was removed, and (d) if the claim in the advertising circular that the product is recognized all over the world as "the best preparation of its kind" was abandoned. In its reply the firm stated that its product has been used in some hundreds of cases without a report of untoward effect; that it is not willing to disclose the details of the "secret process" by which the product is obtained. The firm has not indicated willingness to withdraw the claim "the best preparation of its kind." The Council declared Hepatex P. A. F. unacceptable for New and Non-official Remedies because it is marketed under a proprietary, insufficiently descriptive name without adequate declaration of composition, because there is no adequate evidence for the safety of the recommended intravenous use, and because the claim that the product is "the best preparation of its kind" is unwarranted. (Jour. A. M. A., November 12, 1932, p. 1690.)

PRESIDENT'S LETTER

IT IS with a combined sense of a great responsibility, and a belief in the almost universal approval by the medical men of the state of what the state medical association has done in the past ten years in meeting the problems presented, that I assume the duties of President of our State Association.

Each year presents new problems, and their solution is not a one man job. They call for continued loyalty and support from every quarter of the state.

We may well be proud of the accomplishments of the past ten years—particularly along economic lines. That so great a percentage of effort by the State Association has been given to economic affairs needs no apology or defense. If this had not been true, the individual county societies would be in a sorry plight today; the state would be overrun by the cults, and quackery would be flourishing. Nowhere is the fable of the bundle of sticks better illustrated than in the relationship of the component societies to the state association.

In common with many previously expressed opinions from all over the country, my belief is that the steady overproduction of physicians is the biggest problem we face. Any action on our part cannot settle the questions for the entire country, but Minnesota can start the ball rolling.

It is not reasonable to expect that the initiative in this matter will originate in our medical schools when the final decision rests with the department of administration and the board of regents. The fees from medical students at our university go into the general fund. The physical equipment is designed for a certain number of students—roughly, double the number adequate for the needs of the state. Our population is approaching a stationary point, and in another twenty to thirty years will probably become fixed. And still the flow of young physicians continues. It is obviously unfair to do the weeding process after a man has spent five years in study. The system of the Harvard law school, in which 25 to 30 per cent of the freshman class are weeded out, is a much fairer system. But in the final analysis, a definite quota system of selection of those to be allowed to start the study of medicine seems the most fitting solution of the problem.

The number of Americans studying medicine abroad today is estimated to be as high as 5,000. If all physicians applying for licenses in Minnesota who have received their medical education outside the U. S. were first required to obtain a license to practice medicine in the country in which they received their education, this requirement would eliminate a good percentage. This rule has been adopted by the National Board of Medical Examiners.

We have a committee of men of sound judgment and broad experience who have the investigation of this problem in charge. Let us realize clearly that no selfish and narrow considerations guide us in this matter; the welfare and high standards of the medical profession of the future are at stake.

President,
Minnesota State Medical Association.

F. J. Savage, M.D.
President, Minnesota State Medical Association

EDITORIAL

MINNESOTA MEDICINE

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J. R. BRUCE, Business Manager
324 Fourth Avenue South, Minneapolis, Minn.
and
2642 University Avenue, Saint Paul, Minnesota
Telephones *Dupont 1013* *Nestor 1381*

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All advertisements are received subject to the approval of the Council on Pharmacy and Chemistry of the American Medical Association.

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Vol. XVII January, 1934 No. 1

VITAMIN A DEFICIENCY

As a result of laboratory experiments on animals we have evidently become over-enthusiastic in our reliance on the adding of vitamin A to the diet to prevent respiratory infection. As a result of the clinical trial the subject has been greatly clarified.

In a children's institution, Hess gave cod liver oil, haliver oil, carotene and viosterol to groups of children and showed that none of these diminished the incidence of common colds, pneumonia, skin infections or conjunctivitis and further did not affect the growth of the children. What a welcome discovery for the child that hates cod liver oil.

Animals fed on diet deficient in vitamin A do show a cornification of the mucous membranes and their susceptibility to infection. The assumption seemed warranted that the addition of vitamin A to a child's diet would, therefore, tend to prevent infection. The average child with his susceptibility to acute respiratory infections has no symptoms of vitamin A deficiency.

Hess mentions Wolff's finding that the livers of 60 per cent of newborn babes contained no vitamin A. The incidence of pneumonia in babes under six months however is very low compared to that in the second six months or second year of life—a further indication that vitamin A deficiency has nothing to do with development of pneumonia.

There are certain conditions which doubtless indicate the addition of vitamin A to the diet. A child with xerophthalmia with its susceptibility to pneumonia and other infections simulates the vitamin A deficient animal. A child with a chronic diarrhea, or one on an anomalous diet, an adult suffering from chronic alcoholism or intestinal parasites, or a man such as a coal miner working in unsanitary surroundings conceivably should be benefited by the addition of vitamin A to the diet. Jeans found that 20 per cent of children applying to a certain hospital had diminished acuity of vision in the dark which responded to cod liver oil. In sick children the vitamin A deficiency may be more common than is generally recognized.

The vitamin preparations will continue to be prescribed for indications more clearly definable. It is useless, however, to expect that the addition of vitamin A to the diet will either prevent or cure respiratory or other infections in children who eat well.

RADIO ADVERTISING

It is remarkable how much of radio advertising has to do with health matters. Charlatans, patent medicines, foods, and cosmetics are advertised to the public with not only misleading but crudely false claims. The present state of affairs gives a black eye to legitimate advertising and it is high time something was done about it.

The Copeland bill which is to be submitted to Congress proposes to correct this abuse. It is an extension of the field of the Food and Drug Act of 1906 whereby manufacturers were required to truthfuly label their wares and pro-

hibited from making false and fraudulent claims on patent medicine labels. There is practically universal agreement that this is a valuable law. The Copeland Bill is to extend these requirements to advertising. Advertising of food, drugs and cosmetics shall not be untrue or even misleading in any particular. Trade puffing is still to be allowed as it is felt such language is usually obvious. Drugs further are to be labeled with directions for use which will not endanger health. Perfectly right. Lash-Lure tragedies should be prevented if possible. Labels are to reveal the contents of the package. Even self-medicators are entitled to know what they are taking. A physician insists on knowing what he prescribes. Rightly, the advertiser, not the radio station or newspaper, is to be held responsible.

The objection raised to the Copeland Bill that medical opinion on drugs or remedies is not in agreement applies more to new remedies than to the majority. The secretary of agriculture who will have administrative powers can obtain reliable information on the value of remedies by a questionnaire to reputable physicians, the present method, or by consulting the national headquarters of the profession.

We would like to see a reform in advertising. If we must listen to it over the radio may we be spared the false statements which nauseate.

A letter was recently received by the Hennepin County Tuberculosis Association from Dr. Kendall Emerson, Managing Director of the National Tuberculosis Association, expressing his hearty approval of the campaign recently instituted in Minneapolis aimed at the early detection of cases of tuberculosis. A syringe and tuberculin, with instructions for the application and reading of the Mantoux test, have been recently sent to Minneapolis physicians by the Hennepin County Tuberculosis Association.

COMPULSORY SICKNESS INSURANCE

"Such experience of life as I have had, together with my reading of the experience of others in this sphere, suggests that *we have made a mistake here.* It suggests, further, that *the mistake has been owing to an imperfect attention to the habits and psychological reactions of a significant proportion of working-class families.* What proportion such families as those I am thinking of bear to the whole I do not know—it may easily be as large as one-fifth or a quarter. *They consist of persons who have very little sense of property and who happily have no desire to possess or accumulate it.* They are not necessarily bad or unattractive persons on that account; nor, except at times, are they properly speaking poor. They very likely live in what is described as a poor home, because as a rule there is not much room and very little furniture; but often, and certainly in good times, a surprising amount of money comes into the house each week. *What they have they spend, sometimes on superabundant but often ill-chosen sustenance, almost universally on the more popular forms of enjoyment and entertainment. When times are bad they look for all sorts of windfalls and use all sorts of shifts and stratagems to secure what is needed at the moment.* There is no advantage in including such people in an insurance scheme. They are constitutionally unable to appreciate its meaning. They will resent or fail to pay their contributions. They will honestly regard such cash benefits as the scheme provides for as things to be exploited and by some means or other to be secured when need arises and other resources fail. For them the appropriate means of relieving destitution (not of providing medical attention be it noted—that comes in the other category) is not an insurance scheme but a sympathetically administered Poor Law.

"For the bulk of wage-earners, for that great majority who have a reasonable sense of property and who see the need for thrift, even if some of them cannot understand the niceties of insurance, an insurance scheme to secure cash payments in appropriate circumstances is a most valuable thing. Experience shows that when it is offered advantage will be taken of it. I suggest that while there should be a national scheme for insurance for cash benefits, while there should be extensive propaganda and abundant facilities for such insurance, and within limits governmental contribution towards it, any scheme for these purposes should be on a voluntary basis. It would supply the felt need; no doubt by education and example it would gradually extend its field of recruitment; but it would avoid the difficulties and abuses which most of us have experienced as arising when the opportunity for cash payments is compulsorily extended to those whose ingrained habits and modes of thought prevent them from appreciating the conditions with which those opportunities are surrounded. *I doubt, indeed, whether it will be possible for the nation to continue much longer upon the present lines.*"

EDITOR'S NOTE: The above quoted in the November 1933 *A. M. A. Bulletin* was taken from an article by Sir Henry Brackenbury in the *British Medical Journal*, July 15, 1933. It well expresses the psychological reaction of an appreciable portion of the population in any country to compulsory sickness insurance. Instead of the insurance being a matter of thrift, that portion of the population will demand and get all it can from the government and avoid contributing whenever possible. There are those who think that the present government emergency relief measures are laying a foundation for the expectation, on the part of this same element in our population, of government assistance and medical care as their right permanently.

OBITUARY

Dr. Hartland Cyrus Johnson

Dr. Hartland Cyrus Johnson, prominent Saint Paul physician and surgeon, died Sunday morning, November 26, 1933, after an illness of several months. He was born in New York state in October, 1860.

During his earlier years, Dr. Johnson lived near Northfield, Minn., where he attended school. He was graduated from the College of Physicians and Surgeons of Chicago in 1886, following which he practiced in Farmington several years. He came to Saint Paul in 1888 and practiced here until two years ago, when he retired.

Dr. Johnson was a member of the Ramsey County Medical Society, the Town and Country Club, and Saint Clement's Episcopal Church. He was a Mason and a Shriner.

Survivors are a daughter, Mrs. Harry Oerting, a brother, Dr. Asa M. Johnson; and a granddaughter, Dorothy Marie Oerting, all of Saint Paul.

A FORUM OF THE COMMITTEE ON PUBLIC HEALTH EDUCATION

Federal Emergency Medical Relief

Shall the Government Pay the Doctor?

What is the attitude of the medical profession in general on the subject of Federal Emergency Medical Relief?

No doubt, there is genuine apprehension in some quarters of its possible influence on medical practice of the future.

Everywhere, representatives of organized medicine have proceeded guardedly in making arrangements for the operation of this relief.

The greatest danger to be guarded against, in their opinion, has seemed to be any official sanction for a fee schedule which has been reduced only for emergency care of the indigent.

Accordingly, in Texas, the state society approved no fee schedule at all but arranged for each county society to make its own contract with the state welfare agencies acting as federal administrators of relief.

In Michigan, the state society has suggested a fee schedule, following the lead of Minnesota as to basic rates and reductions, with the specific request to federal authorities, there, that the schedule be kept confidential.

In Minnesota, negotiations with the State Board of Control have been carried on by a group of individuals which included officers of the state society but which was not in any sense an official committee from the society. The suggested fee schedule, with the reduction of 40 per cent arrived at as a result of these negotiations, is therefore in no sense binding upon the membership of the society and each physician is still free to negotiate his own terms with his local representative of the relief administration.

In the three cases cited, no official, standardized fee schedule has been publicly sanctioned by state organizations. In two cases, whose example will probably be followed in many other states, reduction has been made in part by a flat percentage from a basic fee as more desirable than any further explicit reduction of the fee, itself.

The rules and regulations for administration of medical relief as distributed by the Federal Relief Administration are careful to stipulate that there shall be no disturbance of the physician and patient relationship and no control, save the control involved in setting tentative limits to the amount of service to be given without special authorization, is to be exercised over the doctor.

The fact that federal relief funds are now for the first time available to pay the physician's bill along with the grocer's, the coal man's, and the landlord's bill, is obviously gratifying to the profession. This recognition of the right of the physician to remuneration has been long overdue.

The fact that there are people who need immediate emergency medical care and cannot get it, is also obvious and a fact to be faced by the profession. The load has got far beyond what the profession, with the best will in the world, but with credit and charity lists stretched already to the utmost, can carry.

In view of this situation, there seems very little doubt that the vast majority of physicians will accept compensation at the hands of the government for relief cases. Many of them regard it as imperative. The present situation is an emergency which they must meet just as they would meet a war time emergency and without too much regard for problematical consequences at some future time. At any rate requests for information made to the state office indicate a widespread interest in the matter.

For the sake of these inquirers it should be emphasized again, probably, that only those who are already on the relief rolls, and only in counties now receiving federal relief funds, are eligible for this federally financed emergency medical care. The list of those counties, with the names of county relief workers with whom the names of physicians desiring to engage in this type of care must be registered, follows:

Aitkin—Mr. L. Fiske, Aitkin.
Anoka—Miss L. I. Berger, 3977 Van Buren St. N.E., Minneapolis, Minn.
Beltrami—Mr. Ray Carney, Bemidji.
Becker—Mr. Claude Lindquist, Detroit Lakes.
Benton—Miss Mary Starr, Sauk Rapids.
Big Stone—Mrs. Hazel Decker, Ortonville.
Carlton—Miss Margaret Atkinson, Carlton.
Cass—Mr. Glen Holstad, Walker.
Chippewa—Miss Esther Freeman, Montevideo.
Clearwater—Mr. W. Beard, Bagley.
Cook—Miss Ruth C. Ferguson, Two Harbors.
Crow Wing—Miss Mildred Hagstrom, Crosby.
Hubbard—Mr. Maurice Warble, Park Rapids.
Isanti—Miss Laddie McNamara, Cambridge.
Itasca—Mr. R. W. Gifford, Grand Rapids.
Kanabec—Mrs. Mabel Ravenscraft, Ogilvie.
Kittson—Mrs. John Brendal, Hallock.
Koochiching—Miss Ellen M. Saarela, International Falls.
Lake—Miss Ruth C. Ferguson, Two Harbors.
Lake of the Woods—Miss Hilfred Johnson, Baudette.
Mahnomen—Mrs. Otto Isaacson, Mahnomen.
Marshall—Mr. Stephen S. Easter, Warren.
Mille Lacs—Miss Lyle Byrnes, Milaca.
Minneapolis (City)—Mr. M. U. G. Kjorlaug, Division of Public Relief, Court House, Minneapolis.
Norman—Mrs. Eleanor Bue, Ada.
Ottertail—W. I. Prince, Fergus Falls.
Pine—Miss Julia Zupanich, Pine City.
Roseau—Mr. C. S. Rondestvedt, Roseau.
St. Louis Park (City)—Mrs. E. H. Renner, City Worker, 5806 Goodrich Ave., St. Louis Park, Minn.
Staples—Mr. B. C. Barrett, Staples.
Stearns—Mrs. Emma Moynihan, Court House, St. Cloud.
Traverse—Miss Elizabeth Burns, Wheaton.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

MEDICAL BROADCAST FOR THE MONTH

The Minnesota State Medical Association Morning Health Service

The Minnesota State Medical Association broadcasts weekly at 11:15 o'clock every Wednesday morning over Station WCCO, Minneapolis and Saint Paul (810 kilocycles or 370.2 meters).

Speaker: William A. O'Brien, M.D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota.

The program for the month of January will be as follows:

January 3—What Price Health?
January 10—Little's Disease.
January 17—First Aid in Poisoning.
January 24—Mental Health in Childhood.
January 31—Jaw Tumors.

THE STATE MEETING

Several exhibits of especial interest are planned for the 81st Annual Meeting of the Minnesota State Medical Association, to be held at Duluth, July 16, 17, and 18, 1934.

One is the prize winning pneumonitis exhibit prepared by John W. Towey, superintendent of the Pinecrest Sanatorium, Powers, Michigan, awarded the medal at the Milwaukee meeting of the American Medical Association. The exhibit is of especial interest and significance to the medical profession in Minnesota inasmuch as it includes the Minnesota hardwoods that have been found to affect workers, here, also complete x-ray films. Dr. Towey has consented to present a paper on pneumonitis, according to the Committee on Scientific Assembly, now engaged in shaping the program for the three-day Duluth session.

The Diabetes Committee of the State Society, under the chairmanship of Russell M. Wilder, Rochester, will sponsor another exhibit of unusual practical value at this meeting. Several sessions for small groups will be held each day in connection with this exhibit. Talks will be given these small groups, together with special instruction in the administration of insulin and in planning special diets. It is proposed that the series take the form of a postgraduate course in diabetes.

Arrangements are practically complete, the Committee reports also, for a notable cancer exhibit with special demonstration of various new diagnostic methods including transillumination of the breast and the Schiller test.

Another exhibit of especial current interest will show the effects of certain harmful cosmetics, particularly those used for dyeing eyelashes and eyebrows, which have received so much medical attention of late. Arthur J. Cramp, Director of the Bureau of Investigation of the American Medical Association, who prepared the exhibit, will be present himself to talk at the meeting on this subject.

A heart exhibit and demonstration and an interesting symposium on endocrines are to be further features of the meeting.

A program constructed on lines similar to those of the Rochester meeting is being developed, the Committee announces. Considerable time will be devoted again this year to the very successful small group demonstrations that featured both the Saint Paul and Rochester meetings.

There will be separate medical and surgical lecture sessions, many of them under the direction of the special societies and amplified by dry clinics.

It is announced that program time is still available for a few good scientific moving pictures. Anyone who has such pictures should communicate immediately with committee members or with the State Office, 11 West Summit Avenue, Saint Paul.

CAMP RELEASE DISTRICT MEDICAL SOCIETY

Drs. R. K. Ghormley, A. R. Barnes and P. A. O'Leary of Rochester, were the speakers at the annual meeting of the Camp Release District Medical Society, held at Granite Falls, November 16. Dr. Ghormley talked on "Common Fractures," Dr. Barnes on "Coronary Disease" and Dr. O'Leary on "Dermatosis of the Palms." The following officers were elected for the coming year: President, Dr. L. J. Holmberg, Canby; vice president, Dr. M. S. Nelson, Granite Falls; secretary-treasurer, Dr. J. Dordal, Sacred Heart.

WOMAN'S AUXILIARY

President—Mrs. A. A. Passer, Olivia
Chairman Press and Publicity—Mrs. Glen R. Matchan, Minneapolis
Editor—Mrs. S. H. Baxter, Minneapolis

The Women's Auxiliary is pleased to announce that the State President, Mrs. A. A. Passer, is convalescing after an operation and will soon be back at work.

The Women's Auxiliary to the Ramsey County Medical Society opened its year's activities in October with a largely attended tea at the home of Mrs. Arnold Schwyzer. Mrs. John J. Ryan, newly elected president, named the following chairmen of standing committees: Mrs. E. H. Boland, Philanthropy; Mrs. E. V. Goltz, Program; Mrs. W. A. Kennedy, Hospitality; Mrs. Harry B. Zimmermann, Membership; Mrs. A. G. Schulze, Ways and Means; Mrs. L. W. Barry, Publicity; Mrs. C. C. Bell, Telephone; Mrs. Edward Schons, Legislative; Mrs. F. H. Neher, Year Book. Mrs. Harry B. Zimmermann is president-elect; Mrs. Leo Hilger, first vice president; Mrs. Harry Oerting, second vice president; Mrs. Harry Ghent, recording secretary; Mrs. P. Roy, corresponding secretary; Mrs. D. K. Bacon, treasurer, and Mrs. Asa Johnson, auditor.

At this meeting it was voted to donate a loving cup which is to be awarded to the winner in Ramsey County in an essay contest on the subject of "Youth, the Hope in Tuberculosis Control." This contest, which is state-wide among the junior high and senior high school students, will be sponsored by the Minnesota Public Health Association.

The November meeting was held at the home of Mrs. W. C. Rutherford. A lovely musical program was given by local artists, followed by tea. Mrs. W. H. Von der Weyer, who was placed in charge of securing Red Cross memberships in the Lowry Medical Arts Building, reported having secured more than her quota.

The sale of Christmas seals in Saint Paul for the first day, December 4, was in charge of Mrs. Harry Ghent and Mrs. Leo Hilger.

On December 5, the Ways and Means Committee, under the direction of Mrs. Albert G. Schulze, sponsored a Baked Ham dinner, given in the Ramsey County Medical Society Assembly Room, Lowry Medical Arts Building. The proceeds of this dinner will be used to replenish the philanthropic fund.

(Mrs. L. W.) May Barry,
Chairman Publicity.

PROCEEDINGS OF THE MINNESOTA ACADEMY OF MEDICINE

Meeting of November 8, 1933.

The regular monthly meeting of the Minnesota Academy of Medicine was held on Wednesday evening, November 8, 1933, at the Town and Country Club. Dinner was served at 7 o'clock. The meeting was called to order at 8 o'clock by the President, Dr. C. D. Freeman. There were sixty-five members and one guest present.

Minutes of the October meeting were read and approved.

Dr. George D. Head, chairman of the Necrology Committee, read the following Memorial to Dr. John W. Bell, a charter member and former president of the Academy.

JOHN WESLEY BELL, M.D., 1853-1933

The Minnesota Academy of Medicine is again called upon to mourn the passing on of one of its oldest and most revered members, Dr. John Wesley Bell. This pioneer physician of the state was born March 18, 1853, in London, Ohio, the son of Robert J. and Ann Bell. His father was a farmer and his early education was carried on in the public school system of that state. In 1876 he received his medical degree from the Ohio Medical College, following which he pursued postgraduate work in London and in Germany.

Six years later he settled in Minneapolis where he began a career of more than fifty years in the practice of our profession. In 1886 he was elected Professor of the Theory and Practice of Medicine at the Minnesota Hospital College and two years later he was made Professor of Physical Diagnosis and Clinical Medicine at the Medical School of the University of Minnesota and served a period of twenty-eight years in active teaching. He was elected to the Minnesota State Senate from 1891 to 1895. He was appointed a member of the Minneapolis Charter Commission. When the Hennepin County Tuberculosis Sanatorium was established he was chosen a members of its commission and served until his resignation in the year 1919.

During his long medical career he received many honors. He was President of the Hennepin County Medical Society in 1902 and of the Minnesota State Medical Association in 1905. He was a Charter Member of this Academy and was elected its President in 1894. He was a member of the American Gastro-Enterological Association, and was elected an Honorary Member of the Minnesota Society of Internal Medicine.

He was married November 11, 1890, to Kate M. Jones, who, with two sons, Dr. Warren Bell and Robert Bell, survive him. He died May 16, 1933.

These, in brief, are the recorded facts in his long and useful life. But it is not the recorded achievements of life which build enduring memories. A man of quiet personality, unusually modest, considerate of his fellow practitioners to a remarkable degree, beloved by his patients, his memory will live in the hearts of those who have known him long after the deeds of more aggressive men have been forgotten. There are important achievements in his useful life which demand specific notice: his career as a member of our profession and as a consultant, his services in medical education, and his contribution to the literature of his day. It is of these outstanding landmarks that we wish to speak.

Never a robust individual, Dr. Bell carried on a very active practice in medicine over many years. Unremitting in his labors, out early each day, he was strict in keeping his appointments and he made it a matter of professional honor always to be at his consultations on time. Many of those who had been his former pupils looked to him as their consultant and modeled their ways after his.

With unusual clinical powers of observation, an accurate diagnostician, he had a genuine sympathy for his patients and he maintained in his relations with them the highest ideals of courtesy and kindness. His whole life was given over to the interests of medicine. He had other activities but they were of relatively minor importance. At his death, he left behind him countless friends among the rich and poor.

As a member of the original Faculty of the School of Medicine of the University of Minnesota, Dr. Bell is mentioned in the minutes of the Board of Regents' meeting in 1888. He served as Professor of Physical Diagnosis and Diseases of the Chest from 1891 to May 1905, at which time he was made Emeritus Professor of Physical Diagnosis and Clinical Medicine and served as such to the date of his death. His addresses to the Senior students of the Medical School upon medical ethics and medical economics were given subsequent to his retirement from active teaching. As late as one year ago Dr. Bell gave one of these lectures upon Medical Ethics full of practical advice and sound counsel to young practitioners.

It is impossible to estimate the profound influence of this man's lectures upon the young men entering the profession, and Dr. Bell was, both by precept and example, the "Admirable Crichton" of medicine. We, who were at first his students and later his confreres, can only now honor him by striving to reach the heights he so often exhorted us to work toward. Truly, there is a "lonesome place left against the sky" since John Bell died.

His contributions to medical literature are comprised in eighteen bibliographic records which we have been able to find and which we wish to record.

BIBLIOGRAPHY OF THE WRITINGS OF DR. JOHN WESLEY BELL (1853-1933)

Compiled October 16, 1933

A Clinical Lecture on Valvular Lesions of the Heart. Northwestern Lancet, 8:197, 1888.

The Etiology of Phthisis Pulmonalis. Northwestern Lancet, 10:220, 1890.

Placentia Previa, or Placental Presentation. Columbus Med. Jour., 15:245, 1895.

A Plea for the Aged. (Presidential Address before the Minnesota Academy of Medicine, Nov. 7, 1894.) Northwestern Lancet, 15:41, 1895.

The Management of the Senile Heart. Medical News, 69:344, 1896.

The Early Clinical Manifestations of Pulmonary Turberculosis. Northwestern Lancet, 23:57, 1903.

Clinical Aspects of Chronic Myocarditis. Medical News, 84:880, 1904.

Prognosis and Treatment of Chronic Myocarditis. Northwestern Lancet, 24:321, 1904.

Gastric Cancer. St. Paul Med. Jour., 7:510, 1905.

The Need of a More Rational Therapy. Northwestern Lancet, 25:292, 1905.

The Diagnosis and Treatment of Chronic Myocarditis. Jour. Minn. Med. Assn., 26:277, 1906.

The Diagnosis and Treatment of Gastric Ulcer. Jour. Minn. Med. Assn., 27-311, 1907.

The Management of Gastric Ulcer. St. Paul Med. Jour., 9:233, 1907.

Treatment of Gastric Ulcer. Jour. Minn. Med. Assn., 28:281, 1908.

Chronic Myocarditis and Angina Pectoris. Journal Lancet, 39:307, 1919.

Physical Signs of Early Pulmonary Tuberculosis. Minn. Med., 4:382, 1921.

Medical Ethics. Journal Lancet, 47:126, 1927.

Medical Economics. Journal Lancet, 48:237, 1928.

The subjects treated are varied, five are devoted to the heart and its diseases; three to pulmonary tubercu-

losis, three to gastric diseases (cancer and ulcer), and three upon such general topics as medical ethics and medical economics, rational therapeutics, etc. Quotations from some of these writings reveal the character of the man.

In a paper upon "The Early Manifestations of Pulmonary Tuberculosis," he writes: "Pulmonary tuberculosis is emphatically a disease of all times, all countries and all races. No climate, no latitude, no occupation, no combination of favoring circumstances affords perfect immunity. It invades alike the homes of the rich where every comfort abounds and the abodes of the poor where the misery of a hopeless disease is rendered more miserable by the pangs of poverty and neglect."

In a paper on the senile heart he says: "I desire to emphasize first the necessity, yea the obligation, for early and prompt recognition of the danger signals indicative of beginning senile cardiac insufficiency, believing that no practitioner is justified in ignoring or passing as unworthy of the therapeutic attention the breathlessness, precordial distress, and irregularity of the pulse, so often encountered after middle life."

In his presidential address before the State society in 1905, "A Need for More Rational Therapy," he says: "As physicians we should remember that in the eyes of the people treatment is everything, the Alpha and Omega of Medicine." "As a profession we have directed all our efforts and energy to the investigation, detection and prevention of disease, leaving the individual ill with disease to take care of himself." "I know of no man in or out of our ranks so positively harmful and detrimental to the progress of medicine as the therapeutic nihilist."

In his address on medical economics delivered to the Senior class of medicine in 1928, we read: "A professional life may be likened to a road or highway and success its destination. He who keeps on the highway and follows the road signs usually avoids the bumps and saves worry and expense." "Medicine has for its prime object the service it can render humanity but the physician must live in order to render the service." "A physician is a public personage and should manifest an interest in the community."

In his lecture on Medical Ethics, delivered at the Medical School in 1927, we get many sidelights upon Dr. Bell's character. To quote from the lectures: "It is well to remember that more men fail in the practice of medicine from the inability to understand and manage people than from ignorance of the science of medicine, colossal as the latter may be at times." "The future of medicine rests with the students of today." "The physician's life is a strenuous one but its rewards are many and within the reach of all who sincerely seek to serve humanity." "The ability to inspire confidence, natural to some, is a most valuable aid to a physician." Sympathy more than any other quality wins the hearts and confidence of patient and relatives." "The physician should remember that scandal is like an egg—when it is hatched it has wings." "He should remember that the poor are his first and most loyal patients." "No individual carries greater responsibilities to the generation in which he lives or the community in which he practices than the physician." And finally he pronounces his own benediction in these words: "And when life's day is spent and evening is announced by the inelastic step, the dimmed vision, the impaired memory and the hardened arteries, the frail old man returns to his friend of the morning to be relieved and comforted until his eyes are closed in the eternal sleep."

And now we have turned the leaves in his book of life; the last page has been read. We close the volume. Stimulated by his achievements, his wisdom and his faith and example, we fix our eyes upon the future of our beloved profession with hope and courage.

The Committee:
ARTHUR S. HAMILTON,
JOHN E. HYNES,
GEO. DOUGLAS HEAD, *Chairman*

Dr. E. L. Gardner, Chairman of the Necrology Committee, read the following Memorial to Dr. Emil S. Geist, a past president of the Academy.

EMIL S. GEIST, M.D., 1878-1933

Dr. Emil Geist was born in Saint Paul, Minnesota, on May 9, 1878. Following his graduation from the Medical School of the University of Minnesota in 1900, he served one year as an intern at St. Joseph's Hospital in St. Paul and then spent three years doing postgraduate work in Paris, Breslau and Vienna. In Breslau he was associated with the noted surgeon, Mikulicz, at a time when Sauerbruch was associated with the clinic and in Vienna he studied and formed a lasting friendship with Dr. Adolph Lorenz. Upon his return home he entered the practice of orthopedic surgery in Minneapolis, where he was active until his death.

In 1911 he married Miss Augusta Ohage, daughter of Dr. Justus Ohage of St. Paul.

Dr. Geist served as Major in the Medical Corps of the United States Army during the World War, and conducted schools of orthopedic surgery at Fort Oglethorpe, Georgia, and Fort Sam Houston, Texas.

His name will best be remembered for his contributions to Orthopedic Surgery. He was the founder and "Father" of the Minnesota Orthopedic Society, and served as a teacher of orthopedic surgery at the Medical School of the University for many years. He was head of the orthopedic department of the Minneapolis General Hospital for many years, and was on the active staff of all the private hospitals in Minneapolis. He was a very active member of the American Orthopedic Association and he became a charter member of the International Society of Orthopedic Surgery founded in Paris in 1930. His contributions to medical journals were many and important. He was particularly interested in the progress of his specialty and rarely failed to attend the meetings of any important orthopedic society in America or abroad.

He was an active member of the American Medical Association, the American College of Surgeons, the Minnesota State Medical Association, the Hennepin County Medical Society, the Minnesota Pathological Society and the Minnesota Academy of Medicine. In 1928 he served as president of the Hennepin County Medical Society and in 1930 the Minnesota Academy elected him its presiding officer. He was a member of Alpha Kappa Kappa Medical Fraternity and of the Minneapolis Club.

Dr. Geist had an unusually active mind not only in medicine but also in music, art, literature, languages and outdoor life. His main diversion was to invite various members of the Minneapolis Symphony Orchestra to his home for evening dinner and then spend several hours playing chamber music during which he played a score for the violin. His speaking knowledge of European languages broadened his acquaintance and developed a real cosmopolitan viewpoint. Dr. Geist was in unusual demand for holding clinics, not only for his masterly presentations but also because of his genial, whole-hearted nature which made friends in all his contacts. No one ever gave more time to unfortunate physicians and their families or to the poor who could not pay a fee. He never thought of saving himself until he suffered his first coronary attack about five years before his death. To his great credit he was able to adjust his habits and his mental viewpoint in spite of the knowledge his end might come at any time. He on

several occasions had stated that each added year was a gift of fate and that he was enjoying and making the most of each day as it came, without fear of the end.

Death came suddenly on May 14, 1933, at his home a few hours after he returned from a meeting of the American Orthopedic Association in Washington. He is survived by his wife, one son, Justus John, and two daughters, Annamarie and Louise.

The Minnesota Academy of Medicine mourns not only the loss of a loyal member, but also the contact with a whole-hearted stimulating personality.

The Committee:
GUSTAV SCHWYZER,
WALLACE COLE,
E. L. GARDNER, *Chairman*

THE EYE IN CARDIOVASCULAR DISEASE

ARTHUR EDWARD SMITH, M.D.
Minneapolis

ABSTRACT OF INAUGURAL THESIS

Increased incidence of cardiovascular disease in past three decades. Due to greater prevalence of cases of coronary sclerosis-hypertension group.

Reasons for characteristic reaction of the eye tissue to the effects of disease of the heart and blood vessels. Unique opportunities for observation of the presence and progress of vascular pathology in the fundus of the eye.

Classification of diseases of the heart and blood vessels affecting the eye.

Significance of pulsation of the retinal vessels.

Etiology and essential nature of hypertension, with description of fundus changes observed in the various forms of hyperpiesis including the hypertensive toxemia of pregnancy.

"Albuminuric retinitis"—an obsolete and misleading term, since albuminuria has no bearing upon the condition, which is a degenerative rather than an inflammatory process. In retinopathy associated with kidney disease hypertension is the underlying factory which is primary responsible for both the nephritis and the retinal lesions.

Types of kidney disease associated with retinal pathology.

Prognostic significance of hypertensive neuro-retinopathy. Pathologic anatomy of the eye in hyperpiesis.

Arteriosclerotic retinopathy—may exist as a distinct entity or be associated with hypertension. Characteristic fundus picture. Advanced arteriosclerotic changes in the fundus (especially in senile arteriosclerosis) often seen in the presence of normal visual acuity. Pathologic anatomy of arteriosclerotic retinopathy.

Difficulties encountered in the study of the micro-Effects of the various dyscrasias and diseases of the blood affecting the integrity of the capillary walls, with resultant hemorrhage, etc.

Difficulties encountered in the study of the microscopic pathology of the eye. Scarcity of material for examination. Most enucleated eyes are the seat of advanced pathological changes and there are few opportunities for making microscopic examinations of eyes in the earlier stages of disease.

In the future, probably most of the advancement of our knowledge in regard to the eye pathology in cardiovascular disease will be achieved through improved methods of clinical examination with the slit lamp, the newer types of magnifying ophthalmoscopes, employment of short-wave-length light, etc.

SOME EXPERIENCES IN SURGERY OF THE COLON

A. R. COLVIN, M.D.
Saint Paul

DISCUSSION

DR. A. E. WILCOX (Minneapolis): Dr. Colvin has presented this paper in his usual interesting and instructive manner. Regarding the diagnosis in acute conditions of the colon, I presume we will always have more or less of the same difficulty; and these difficulties he has emphasized in his remarks and recital of the case histories. After the pathology is definitely established upon exploration, an analysis of what appeared to be trivial symptoms often becomes illuminating.

Carcinoma so seldom gives any suggestive symptoms, particularly of obstruction, until the impaired lumen of the bowel suddenly becomes blocked by some fecal impaction or fruit seeds, that no complaint is made by the patient and surgical contact is delayed until these acute abdominal conditions arise.

What I wish to emphasize particularly in the consideration of Dr. Colvin's paper is the preoperative preparation of the patient. The nasal suction apparatus as devised by Dr. Owen Wangensteen is an admirable means of decompressing the abdomen and getting these cases of obstruction in condition for operative procedure. This method has been presented before this Academy and elsewhere by Dr. Wangensteen. We have, since he familiarized us with this apparatus, been much impressed by its usefulness and efficiency in cases of obstruction and have obtained results which we believe make it preferable to cecostomy. I cannot speak too highly of this preoperative preparation of the patient by the nasal suction method.

DR. H. B. SWEETSER (Minneapolis): I wish to compliment Dr. Colvin on his exhaustive and very instructive paper on diseases of the colon and their treatment. In his report of a case of cancer of the sigmoid he stated that he resected the tumor and reunited the intestinal tube, but that a year afterwards there was a recurrence with numerous metastases. It ocurs to me that in our effort to make the postoperative life of our patients not too uncomfortable and inconvenient, we are liable to be too conservative in our surgical attack.

In this connection I would like to report a case I operated in November, 1932, one year ago. The patient had an acute intestinal obstruction due to a carcinoma of the sigmoid, low down. I resected the sigmoid, from about six inches above the anus to six inches above the tumor, and implanted the proximal end into the skin, through the split fibers of the rectus muscle as an artificial anus. This man is so far perfectly well and at work daily. His bowels move daily in the morning before he goes to business; he is not troubled with expulsion of flatus, and there is practically no (or little) soiling of his bandage with fecal discharge.

I would like to ask Dr. Wilcox if he depends entirely on the nasal suction to relieve the intestinal distention without a subsequent colostomy, and if it always succeeds.

DR. A. E. BENJAMIN (Minneapolis): I want to congratulate Dr. Colvin on his paper; there is much food for thought in it. If one examines all these patients beyond 50 years of age by means of x-rays of the colon, I think he will find a large percentage of them will have diverticula. We know that diverticula can occur anywhere from the esophagus down to the anus. Many fistulæ and sinuses from a diverticulitis in the anus occur in these cases. If you will take these x-rays you will not advocate these large enemata with diverticula. I believe many people do themselves much harm by taking large enemata. If these patients use oil enemata and are put on liquid or semi-solid diets, some of them

will improve. I have a number of patients with diverticula of the colon at the present time who are feeling better. Very few of them will require operation excepting the untreated ones may develop acute attacks. Of course, some of these will have to be operated. It is difficult to cure and many patients develop obstructions. To make a diagnosis between diverticulitis and appendicitis occasionally is rather difficult.

I think a primary colostomy should be done in nearly all of these cases where there is complete obstruction. I have used the nasal suction, as Dr. Wilcox mentioned, before and after operation. After a carcinoma is removed and an anastomosis is performed, it is best that a small opening be left at the site of the colostomy. I do it in such a way that a small vent is left in the bowel for the leaking of gas from the bowel for weeks afterwards. This relieves the patients of gas distention and suture tension and prevents a blow-out. I think this is a very interesting subject.

Dr. Gustav Schwyzer (Minneapolis): I would like to mention a patient whom I operated upon seven years ago next January. She was 30 years old at that time. I operated upon her first for an intestinal trouble that looked like appendicitis. There was no blood in the stool, very slight tenderness of the abdomen, but this slight painfulness more on the right hand side. The bowel movements were normal. Seven months later she came to the office during my absence and my associates found her to have a large tumor in the abdomen, slightly movable, and not markedly painful to touch. As no symptoms of obstruction were existing, the possibility of a foreign body was thought of. On my return in the fall I found, on my examination of the patient, a well-demarcated, large tumor, of irregular form, in the region of the navel. The hemoglobin was reduced to 38 per cent. I excluded the possibility of a foreign body on account of now discovering blood in the stool. It became probable that we had to do with malignancy. Repeated gastro-intestinal x-ray examinations finally brought out a marked defect in the transverse colon, which findings confirmed our diagnosis. After giving the patient 600 c.c. of citrated blood that brought the hemoglobin up to 44 per cent, we incised the abdomen, this time in the mid-line, and found a large carcinoma of the transverse colon, 12 cm. long and 5 cm. thick. I made an end-to-end anastomosis after resecting the cancer of the transverse colon, reuniting the two ends without any tension, because the splenic and hepatic flexura coli were freed, according to Moynihan. The patient left the hospital in two weeks. She never vomited after the operation and is comfortable today.

I want to mention this case for the reason that it is rare to find such large adenocarcinoma of the transverse colon in a young person. Dr. Benjamin made the statement that the colostomy would be the safest procedure and should be done first. I think his thought is correct in all cases of obstruction, but the ideal way is the one-stage operation with an end-to-end anastomosis if conditions present themselves feasible for this method.

R. T. LaVake, M.D., *Secretary.*

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